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DISEASES OF THE LYMPHOID TISSUE OF THE CONJUNCTIVA.*

By E. TREACHER COLLINS.

A PAPER dealing with diseases of the lymphoid tissue of the conjunctiva might readily lead to a discussion of all the inflammatory affections of that membrane, for in all of them the fibro-adenoid layer becomes implicated. It is, however, mainly diseases which are characterised by marked over-development of lymphoid tissue which I propose, in opening this discussion, to describe, and especially the most serious of these—trachoma.

Trachoma is a disease of such exceedingly long duration that few observers have the opportunity of watching cases from the commencement to the finish. In this respect I have had exceptional advantages. The Metropolitan Asylums Board has established Ophthalmia Schools for the isolation of children who, while under the charge of the Poor Law Authorities in London, are found to be suffering from any contagious eye disease. The number of children admitted to these schools in the past five years, during which time I have held the appointment of Ophthalmic Surgeon to them, has been 2441, of which 700 were suffer-

* A paper read in opening a discussion before the British Medical Association, July, 1909, on Diseases of the Lymphoid Tissue of the Conjunctiva.

ing from trachoma. In many of these cases the disease has been in an exceedingly early stage. The children have been sent for admission, not because they made any complaint of their eyes, but because the medical officers of their parish schools noticed some abnormal discharge from them. The children when admitted to the Ophthalmia Schools are kept there, unless their parents insist on taking them out, until they can be certified as cured. It will be seen, then, that these schools afford excellent advantages for the study of the natural history and treatment of trachoma. It is the outcome of the experience which I have gained at them that I now propose to lay before you.

That trachoma is a contagious disease has been proved by inoculation experiments. I have had, however, some very definite confirmatory evidence. When first one of these ophthalmia schools was started, a "house mother" and an assistant nurse employed there contracted trachoma. In one case there was the history of some water having splashed into the woman's eye while she was engaged in washing a child who had trachoma, and it was from that time that the onset of the affection dated.

The way in which the disease started in these two cases is of interest, because they were seen from the very commencement. In both, the symptoms at first resembled those of an acute mucopurulent ophthalmia. There was swelling and redness of both ocular and palpebral conjunctiva, some papillary enlargement of the latter, and mucopurulent discharge. The symptoms, however, did not yield to treatment in the satisfactory way which is usual in that affection, and when the congestion of the palpebral conjunctiva had somewhat subsided the typical lymphoid follicles in the tarsal conjunctiva and the retrotarsal folds became apparent. In one case the affection remained confined to one eye, in the other both eyes ultimately became involved. Both cases were cured after about eighteen months' treatment.

The Ophthalmia Schools are arranged in what is known as the cottage system. They are situated, one at Swanley, in

Kent, and the other at Brentwood, in Essex, and have large and beautiful grounds. Some of the children when their eyes are cured are very loth to depart from these pleasant surroundings to return to their parish barrack schools in London. Various tricks are played by them to keep up discharge from their eyes, or to make them injected, so that they may not be sent away. One child, whose name peculiarly enough was Sly, was admitted with marginal blepharitis and slight conjunctivitis; this was cured and all treatment was stopped preparatory to her leaving the school. She was then caught by the nurse taking discharge from another child's eye who was suffering with trachoma and deliberately putting it into her own. The symptoms of acute mucopurulent ophthalmia rapidly set in, and two months later typical trachoma follicles on the tarsal conjunctiva were recognised. After a year's treatment the trachoma was cured. The contagious character of trachoma being definitely established, there can be little doubt that the contagious element is some micro-organism.

During the last two years, Prowazek and Greeff, working independently, have discovered in the cells from the trachomatous conjunctiva, by the use of Giemsa staining methods, minute slightly ovoid bodies considerably smaller than the smallest of the known cocci. They are found massed together in the cells near the nucleus in the form of a cap, but separated from it by a clear space. The enclosure containing the granules enlarges rapidly, causing the cell to swell and ultimately burst, the granules then being discharged. So far these parasites, if such they are, have not been cultivated or made to reproduce the disease, the evidence in favour of their being the specific organism of trachoma resting on the frequency with which they have been found in cases of that disease, and in the failure to find them in other affections.

Whatever may be the contagious element of trachoma, we may feel sure that it is one which is not spread through the air; that it is, like many of the other organisms which

give rise to contagious ophthalmia, easily killed by drying, and not spore-producing.

Individuals may live surrounded by those suffering from the disease and by the observance of a few simple precautions never become affected. At the Ophthalmia Schools there are employed a large staff of nurses, teachers, and attendants, but the two cases referred to are the only ones of trachoma which have arisen amongst them. If the contagion could be carried in the form of dust in the air, more of the attendants, or I myself who look closely into all the children's eyes every ten days, should surely have become affected.

In countries such as Egypt, where the majority of the native population suffer from the disease or its sequelæ, the Europeans who visit the country and observe precautions as regards cleanliness, which are quite foreign to the natives, rarely contract it. All the evidence, I think, tends to show that it is the transference of moist discharge from one person's eye to another which disseminates the disease.

In some diseases of the conjunctiva, due to microbial infection, *e.g.*, gonorrhoeal ophthalmia and diplobacillary ophthalmia, the organism is found chiefly in the epithelium. In others, *e.g.*, tuberculosis, the organism is located, not in the epithelium but in the sub-epithelial tissue. A comparison of the histological characters of a tubercular nodule of the conjunctiva with those of a trachoma follicle would suggest that the position in which the trachoma organism is usually situated is, like that of the tubercle bacillus, the fibro-adenoid layer.

A tubercle nodule and a trachoma follicle both consist of an aggregation of cells, the most peripheral of which are small lymphocytes and the more central of the epithelioid type. In the centre of a tubercle nodule is the characteristic giant cell, with peripheral arranged nuclei and degenerate cytoplasm, in which the tubercle bacilli are found. In the centre of a trachoma follicle there is no giant cell, but the epithelioid cells are there most degenerate. It would seem,

therefore, to be the position where the toxine is most intense and where the organism generating that toxine would most likely be situated.

Mayou* has further pointed out that in a trachoma follicle plasma cells are absent, though occasionally found in its outer zone. As there is good evidence to show that plasma cells are not very resistant to toxins this arrangement of them is further evidence that the intensity of the toxine is greatest in the centre of the follicle.

Prowazek insists that the ovoid bodies which he has found in cases of trachoma are met with only in the epithelial not in the lymphoid cells. Greeff states that after a case has been treated with sulphate of copper these bodies disappear from the superficial cells and the secretion long before the case is cured. He suggests that they pass into the deeper parts where they can still excite changes.

Some micro-organisms we know, like the gonococcus, can attack the intact epithelium of a mucous surface. Whether or not the organism of trachoma is able to do this cannot yet be determined. The not uncommon accompaniment of trachoma in its early stages with an acute conjunctival inflammation, which is found to be due to Koch-Weeks bacillus or the gonococcus, suggests that breaks in the continuity of the epithelium, or softening of it, may facilitate the passage of the trachoma organism into the adenoid layer; a mixed infection of the two diseases having been implanted at the same time.

Wherever the trachoma organism is located the changes which it or its toxins excite are most marked in the adenoid layer of the tarsal conjunctiva and the retrotarsal folds, *i.e.* in those parts of the conjunctiva over which the epithelium is thinnest.

Most observers speak of the upper retrotarsal fold as the part which is generally first affected. Having seen, as already stated, many very early cases of the disease, I am inclined to consider the appearance of grey, scattered,

* Changes produced by Inflammation in the Conjunctiva, 1905, p. 116.

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avascular spots, rather smaller than a pin's head, in the tarsal conjunctival of the upper lid, as quite as early a symptom as the presence of follicular enlargements in the upper retrotarsal fold.

These grey spots have been termed "elementary or primary granulations" (v. Graefe, Jacobson). Their appearance is quite characteristic and of considerable diagnostic importance. Their presence, when there is much mucopurulent ophthalmia in association with the trachoma, and consequently considerable congestion and papillary enlargement, is often obscured from view, and it is not until these other symptoms have subsided that they become visible.

There are some microbial diseases of the conjunctiva, like gonococcal infection and Koch-Weeks bacillus infection, in which the chief effect of the toxin is the outpouring of a large number of polymorphonuclear leucocytes which infiltrate the tissue and form the predominant cellular element in the discharge. These leucocytes have marked phagocytic powers, and the organisms may be found in the cells in various stages of digestion.

In another microbial disease, viz., diplobacillary conjunctivitis, Stock, Mayou,* and Brown Pusey† have shown that there is little or no increase in either polynuclear or mononuclear leucocytes, but a large increased growth of epithelium, and the formation immediately beneath the epithelium of a number of plasma cells. The discharge is not purulent but consists of mucin, from increased activity of the epithelium, degenerate epithelial cells and the bacilli.

The trachoma organism would also appear to be a non-pyogenic one, the reaction which is excited by its toxine being an immense new formation of lymphoid tissue, a large increase of plasma cells, and in the latter stages a formation of fibroblasts. Though in many cases of trachoma some polynuclear leucocytes are found in the discharge, they do not appear to be a consistent element, and when present can

* Changes produced by Inflammation in the Conjunctiva, 1905, p. 102.

† Archives of Ophth., vol. xxxviii, No. 1, 1909, p. 8.

usually be attributed to infection with other organisms or the presence of other chemiotactic substance. Mayou has pointed out that a characteristic of smear preparations obtained from trachomatous conjunctiva by gentle friction is the presence of an enormous number of plasma cells.

Trachoma *per se* is essentially a chronic disease, the acute symptoms which sometimes usher in an attack, or which arise from time to time in the course of it, can always be accounted for by mixed infection. In the trachoma cases admitted to the Ophthalmia Schools I have frequently found organisms in the discharge, such as Koch-Weeks bacillus and staphylococci, and when by appropriate treatment these have been eliminated, the trachoma has remained as a chronic disease. In these Schools, where the children's eyes are regularly bathed and attended to several times a day by skilled attendants, acute exacerbations of symptoms are very uncommon.

Koch, while in Egypt in 1883, found in the conjunctival discharge in trachoma cases two different kinds of micro-organisms, the gonococcus and what is now known as the Koch-Weeks bacillus. As neither of these is the specific organism of trachoma, the cases must be regarded as having had a mixed infection.

The descriptions which are given of the outbreak of ophthalmia in this country in 1801 and 1802 as the result of the return of Sir Ralph Abercromby's troops from Egypt, is that of an acute purulent ophthalmia with rapid destructive ulceration of the cornea. Some of the cases later on, however, presented the characteristic cicatricial contraction and pannus of trachoma. I think that there can be little doubt that these cases were of a mixed character, of gonorrhoeal ophthalmia plus trachoma.

I think it is incorrect and misleading to speak, as so many of the text-books do, of two types of trachoma, a papillary type and a follicular type. The essential element of trachoma is the follicle; it is the characteristic form of reaction on the part of the tissue to the irritation of the

trachoma organism. Papillary formation over the tarsal conjunctiva, on the other hand, is present in many acute and chronic inflammatory affections. It may, it is true, exist with the follicles in trachoma and sometimes be so extensive as to obscure them, but without the presence of lymphoid follicles we have no right to diagnose a case as one of trachoma.

A characteristic of the trachoma follicles is their tendency to become confluent and produce bodies resembling in shape the grains of boiled sago. This running together of the follicles is most met with in the loosely attached tissue of the retrotarsal folds, but may occur in severe and more advanced cases in the more firmly attached tarsal conjunctiva. As the follicles increase in size the epithelium overlying them becomes thinned and the central part disintegrates until ultimately they rupture, gelatinous contents escaping. On everting the lids in an advanced case of trachoma, many follicles may often be seen just on the point of rupturing and with a very little pressure their contents can be extruded.

Microscopical examination of a follicle which has been ruptured by expression shows (as pointed out by Mayou) invasion of its walls and cavity by polynuclear leucocytes, due, he suggests, to the presence of septic organisms from the conjunctival sac, or possibly I would suggest to chemio-tactic action set up by products eliminated from the damaged epithelial cells.

It has been long recognised that chronic cases of trachoma may be considerably benefited by an acute inflammatory attack. Microscopically it has been found that the trachoma follicles may become invaded from the periphery by polynuclear leucocytes. Such cells do not form part of what may be considered the normal constituents of a trachoma follicle. When, however, an exudation of these highly phagocytic cells is brought about by pyogenic organisms, or the application to the conjunctiva of some irritant such as jequirity, or sulphate of copper, they will invade the follicles and effect their absorption.

Besides the follicular formation in trachoma there is a considerable formation of plasma cells immediately beneath the epithelium between and beneath the follicles; also in the later stages a formation of fibroblasts. I do not propose to enter into the vexed question as to the origin of plasma cells: every possible origin has found its advocate who has supported his view for more or less plausible reasons. Their ultimate fate is also to some extent a moot point, there being a difference of opinion as to whether or not they are capable of developing into fibrous tissue. In the later stages of some cases plasma cells may undoubtedly break down and produce a hyaline material which gives rise to the condition known clinically as "Stellwag's brawny œdema." In its portions, sometimes large areas, of the palpebral conjunctiva have a smooth surface and waxy gelatinous appearance. This condition is similar to, though more extensive than, the milky, opalescent, appearance of the surface of the conjunctiva met with in spring catarrh, which is produced, not as was at one time thought by a thickening of epithelium, but by a hyaline degeneration of plasma cells immediately beneath the epithelium.

The fibrous tissue which forms in trachoma is probably mainly developed from fibroblasts derived from connective tissue cells. It makes its appearance either as strands and irregular networks, or over a large area, which loses its transparency and becomes a dull yellowish white colour. The formation of fibrous tissue and of areas of hyaline degeneration are often associated in the later stages of the disease. The fibrous tissue as it forms replaces the adenoid layer; it destroys the trachoma follicles, partly by forcing them to the surface, where they rupture, and partly by compressing them, and in its contraction causing them to shrink. Some small superficial scar areas are no doubt formed at the seat of the ruptured follicles, but the most extensive growth of fibrous tissue begins in the deeper layers of the membrane and extends forwards to the epithelium.

It is possible for very extensive replacement of the

adenoid layer by fibrous tissue to take place without much distortion of the lid being produced. It is the formation of fibrous tissue in the form of a longitudinal band along the line of the sulcus subtarsalis (Arlt's Scar streak) which most frequently gives rise to entropion and boat-shaped lid.

It is worthy of note that, in cases where there has been extensive formation of fibrous tissue, the condition of the conjunctiva comes to resemble very closely that of the skin. The palpebral conjunctiva differs from skin in the following three particulars:—

(i) The thinness of the epithelium. Over the tarsus the epithelium of the conjunctiva has only two layers of cells; there is no prickle layer and no keratinised layer.

(ii) The absence of papillæ.

(iii) The presence of the adenoid layer immediately beneath the epithelium instead of the dense fibrous tissue of the corium.

The reason for the reduction of the epithelium of the tarsal conjunctiva to such extreme thinness is, I would suggest, to allow the blood in the rich vascular plexus of the adenoid layer to impart warmth to the avascular cornea when the lids are closed. The absence of papillæ allows the smooth working of the two surfaces of the conjunctiva in contact with one another with a minimum amount of friction, which is further facilitated by the mucus secreted from the rupture of the goblet cells.

The thinness of the epithelium of the conjunctiva exposes it to greater risk of microbial infection than the skin. The lymphoid tissue, or the potentiality for the formation of lymphoid tissue, immediately beneath the epithelium, limits the risk of general systemic involvement from any such infection.

In severe and long standing cases of trachoma, fibrous tissue is formed immediately beneath the epithelium of the palpebral conjunctiva, similar to that of the corium of the skin, which like it is raised into papillæ. Instead of being

composed of only two layers of cells the epithelium is thickened into several layers, the surface ones being flattened and squamous; it also sends down numerous finger-like processes between the papillæ. In some cases these processes are converted into gland-like structures, the orifices of which may become occluded, small retention cysts being formed. These little retention cysts are seen clinically as small yellow-coloured patches about the size of a pin's head. If from the contraction of the new-formed fibrous tissue the ducts of the lacrymal and other conjunctival glands become obliterated, so that the membrane is no longer kept moist, then its resemblance to skin becomes still more marked, keratinised layers of epithelial cells being formed on the surface, and the condition termed xerosis being established.

It is frequently asserted that all cases of trachoma result in the formation of fibrous tissue, or cicatrisation as it is termed. The formation of fibrous tissue is, however, one of the later phenomena of the disease; and it would seem reasonable to suppose that if cases are treated sufficiently early they might sometimes be cured without its occurrence. When follicles come to the surface and rupture, whether spontaneously or as the result of mechanical expression, a small scar is probably formed at the seat of rupture. Scars thus formed may, however, be so infinitesimally small as to be quite invisible clinically. "The elementary or primary granulations" met with in the tarsal conjunctiva may, I am quite sure, as the result of treatment, become absorbed and disappear without leaving any trace of previous existence behind. I have seen several cases treated at the Ophthalmia Schools for characteristic trachoma follicles in the tarsal conjunctiva and retrotarsal folds, which, on leaving, have shown no sign of fibrous tissue formation sufficient to have revealed the fact that they had suffered from the disease.

The use of the term cicatrisation for the new formation of fibrous tissue which develops deeply and replaces the adenoid layer is not, I think, a very appropriate one; it would, I suggest, be better termed a sclerosis.

There has been much discussion as to whether the vascular affection of cornea which is met with in trachoma, termed "pannus," is the result of irritation from a roughened eyelid or a real infection of the cornea with trachoma. I would suggest that it is the outcome of both.

Raehlmann* has found pathologically in the anterior layers of a cornea affected with pannus, lymphoid follicles having the same characteristics as those met with in the lids in trachoma. Mayou has also found them in the conjunctiva removed from the limbus by the operation of peritomy for the relief of pannus.

The conjunctiva covering the sclerotic rarely becomes affected with trachoma except at the corneal margin. The question then arises, why should the bulbar conjunctiva be freer from infection than the tarsal conjunctiva, the retro-tarsal folds, and cornea? It might be suggested that the tarsal conjunctiva and retrotarsal folds become affected because they contain lymphoid tissue, and that the bulbar conjunctiva escapes because it does not. If this is so, why, then, should not the cornea also escape, which is most certainly devoid of any lymphoid tissue? If, on the other hand, it is argued that it is the thin covering of epithelium on the tarsal conjunctiva and in the retrotarsal folds which predisposes them to infection, why should the limbus and the cornea, which has thicker laminated epithelium than the bulbar conjunctiva, be more frequently affected than the bulbar conjunctiva? I think an explanation may be found in the damage which must be inflicted to the pliable corneal epithelium by the friction of a roughened lid or in-turning eyelashes over it. In places some of its cells will become brushed away and irregularities will be formed. Into such abrasions the infective trachomatous material becomes subsequently rubbed by the friction of the lids, and so breaks through the protection which the surface epithelium always affords the cornea from external influences.

The formation of lymphoid tissue in a structure like the

* Archiv für Ophthl., Bd. xxiii, 1887, Ab. 3.

cornea, devoid of any normal lymphoid tissue, serves to show that a trachoma follicle is not a mere hypertrophy of pre-existing lymphoid tissue; but that it is a new production for the purpose of defence against the attacking organism.

A study of the natural history of trachoma, as above described, serves to show that the lymphoid follicles which form in connection with it are eliminated in three different ways:

(a) By rupture of the follicles on the surface and discharge of their contents.

(b) By intercurrent inflammation causing follicles to become invaded with polynuclear leucocytes and absorbed.

(c) By the replacement of the adenoid layer by fibrous tissue, which, cutting off the blood supply to the follicles, causes them to atrophy.

Of the immense number of different treatments which have been employed for trachoma, those which have proved most efficacious can, I think, be shown to aid in getting rid of the lymphoid tissue in one of the three above-mentioned ways.

In the operation of expression a large number of follicles are ruptured, and their contents evacuated. If, immediately after expression, the surface of the ruptured follicles is painted with a 1-in-100 solution of perchloride of mercury, an exudate into them of polynuclear phagocytic leucocytes is excited which promotes absorption of the remains of the follicles.

In the operation of expression I prefer to use the flat Grady's forceps rather than Knapp's roller forceps. With the latter I find, unless exactly the right amount of pressure is made, there is a tendency to tear the tissue. My endeavour always is to squeeze, and not to tear. I find a pair of forceps similar to Grady's, which Mr. Tyrrell has had made with a more acute bend, exceedingly useful in catching hold of the upper retrotarsal fold at the inner angle, a position in which a number of follicles are frequently met with.

In performing the operation I employ two pairs of expression forceps, holding the upper lid everted with one pair, and with the other grasping the retrotarsal fold and squeezing the follicles out of it. In the tarsal conjunctiva it is frequently impossible to get rid of the follicles by expression, owing to its firm attachment to the tissue beneath. The so-called "elementary or primary granulations" cannot be squeezed out of the tarsal conjunctiva. To get rid of all the lymphoid follicles from the retrotarsal fold it is often found necessary to repeat the procedure of expression at several different sittings. Even when all follicles seem to have been completely removed from the retrotarsal fold, if the disease is not cured in other parts of the conjunctiva, fresh ones make their appearance which in their turn have to be expressed.

The operation devised by Galezowski of excision of the retrotarsal folds is a very effectual way of getting rid of the follicles and shortening the duration of the disease. I have performed it several times, in cases where there has been much redundance of tissue behind the tarsus. In some cases I have removed all four retrotarsal folds with satisfactory results.

Kuhnt's operation of removal of the tarsus and conjunctiva which covers it I have not performed, not having met at the Ophthalmia Schools with cases which I thought sufficiently severe to justify the employment of so radical a procedure.

Time-honoured sulphate of copper still, I think, maintains the foremost place as the best application in trachoma to stimulate the absorption of follicles which cannot be removed by expression. The reason why a copper salt proves specially useful for this purpose is, I think, to be found in the experiments of Leber. He introduced into the anterior chambers of the eyes of rabbits sterilised substances of different chemical characters, and found different kinds of reaction followed. Thus pieces of gold and glass excited no inflammation, slowly becoming encapsuled by a delicate

connective tissue. Pieces of copper or drops of quicksilver, introduced in precisely similar manner, gave rise to a localised suppuration; whilst croton oil and cantharides caused no pus formation, but a fibrinous exudate with necrosis of tissue.

Copper or its salts then applied to the conjunctiva in a similar way probably set up a mild aseptic purulent inflammation, *i.e.* an exudate of polynuclear leucocytes which will invade the trachoma follicles and tend to bring about their absorption.

In several cases I have endeavoured to increase the efficacy of the copper treatment by forcing it into the tissue by kataphoresis. For this purpose I have used a copper terminal which, with the intervention of a pad of cotton wool saturated with a 2-per-cent. copper sulphate solution, has been applied to the cocainised conjunctival surface of the everted lid. The strength of the current employed has been from 3 to 4 M.A., and the duration of the application varied from 2 to 5 minutes. The process is a decidedly painful one, and is followed by considerable hyperæmia. It is more painful than the application of the sulphate of copper stick, and I cannot say that so far I have found it any more efficacious. I have also, at the suggestion of Dr. Lewis Jones, tried treating the follicles in trachoma by puncturing them with a copper needle, and then passing through it a current of 2 to 3 M.A. for one minute. The hæmorrhage and hyperæmia occasioned prevent more than one or two follicles being dealt with satisfactorily at one sitting, but their absorption certainly seems to be promoted.

Wirtz,* in an article on "Ionic Therapeutics in Ophthalmology," says that in trachoma he has used a $\frac{1}{2}$ -per-cent. solution of copper sulphate with 2 to 3 M.A. for 2 to 3 minutes every few days, and that in four acute cases, after four weeks, the conjunctiva was nearly normal, and four

* Klinisch. Monatsblat. f. Augenheilk., November—December, 1908, and Ophthalmic Review, May, 1909, p. 149.

chronic cases were discharged cured "as far as one can speak of cure in trachoma."

In former times a more violent reaction than that excited by sulphate of copper was effected by the inoculation of gonorrhœal matter. Mackenzie,* in writing of this practice, says: "Although it by no means appears a very safe one, it is undeniable that cures have been effected of the hypertrophied state of the conjunctiva, with the vasculo-nebulous condition of the cornea depending on it." The immense exudation of phagocytic polynuclear leucocytes which was in this way excited doubtless led to the invasion and disappearance of many of the follicles. It can be easily understood that in a tissue normally avascular like the cornea an increase in leucocytosis would be specially useful in promoting absorption of the lymphoid material.

On the introduction of jequirity the use of gonorrhœal matter with its uncertain amount of reaction was abandoned. Now with Merck's extract of abrin, which has received the name "Jequiritol," we have a most efficacious means of exciting, by a chemical irritant, an acute inflammatory attack of limited duration. I have frequently employed it in cases with pannus with good effect. One application of Merck's strongest jequiritol usually suffices to produce the desirable amount of reaction. I have never found it to be so excessive as to necessitate the use of the anti-abrinic serum.

The X-ray treatment of trachoma has the advantage over all other methods of treatment in being painless. It was first introduced by Mayou in 1902, who, in writing of it, says†: "In the X-rays we have a method of setting up a leucocytosis with the absolute minimum of destruction of epithelial or other tissues; and, further, we have a means of producing an inflammation varying from a very slight leucocytosis to an actual gangrene of the part which, with due care and experience, we have under almost perfect control."

At the Ophthalmia Schools the X-ray treatment has

* Diseases of the Eye, 4th edit., p. 647.

† Changes produced by Inflammation in the Conjunctiva, p. 140.

been largely employed for trachoma with variable results. The method of application has been, first to keep the upper eyelid everted with a clamp holding the eyelashes and stuck with a strip of plaster to the forehead. This avoids exposure of the operator's fingers in any way to the rays. The patient is then seated a foot distant from the kathode of the Crooks tube and a five minutes' exposure is made with a current of 10 volts. These exposures are repeated each day for 10 days, and are then stopped for 10 days; if at the end of that time little or no reaction has been set up, application for another 10 days is made.

Some cases treated in this way have steadily improved, follicles disappearing and pannus, when present, clearing up. Some, on the other hand, after very prolonged use of the rays, have shown but little amelioration. In no case have any disagreeable consequences been occasioned from the employment of X-rays in the cautious way above described. The varying results are, doubtless, to a great extent, attributable to varying degrees of dosage according to the hardness or softness of the tube employed. It would be very advantageous if some method for regulating the dosage in the X-ray treatment of trachoma could be introduced similar to the use of the Sabourand and Noiré pastilles in the treatment of ringworm by X-rays.

In 1904 I tried treating cases of trachoma with radium. A 5-milligramme glass tube of radium bromide of low radio-activity was held over the everted eyelids for five to seven minutes daily. This was continued for from 3 to 10 weeks before any definite reaction ensued. The results were not very encouraging. Probably with the improved apparatus for the application of radium introduced by Drs. Wickham and Degais, and longer exposures, more satisfactory effects might be obtained. Success in the treatment of trachoma by radium has been reported by Cohn,* Thieleman,† and Selenkowsky.‡

* Berliner Klin. Wochenschr., No. 1, 1905.

† Zeitschr. f. Augenh., December, 1905.

‡ Arch. f. Augenh., Bd. lx, 1908, H. 1, s. 63.

The third method by which a natural cure of trachoma is effected is, as already stated, the replacement of the adenoid layer by fibrous tissue. I would suggest that some of the more violent mechanical methods of treating trachoma which have been attended by good results act to a large extent by stimulating this natural tendency to fibrous tissue formation. The methods I refer to are those which may be included under the comprehensive term "grattage," whether carried out with a hard brush, a spiked iron instrument, or a knife blade.

I may conclude the description of my experience of the treatment of trachoma by saying that I have found it possible to so thoroughly eradicate the disease that it shows no tendency to relapse. In cases where a relapse seems to have taken place I believe the disease was never completely cured, or that some other inflammatory conjunctival infection (such as that due to Morax's diplobacillus) has become implanted in an eye showing the fibrous tissue sequelæ of trachoma.

I always mistrust statements as to the cure of trachoma in the course of a few weeks. Rapid and striking improvement in some cases is often made by treatment in the first weeks, but the complete cure of the disease, in my experience, always takes several months and sometimes years, whatever procedure be adopted.

I do not consider any case as satisfactorily cured so long as any follicular enlargements can be detected in any part of the conjunctiva, and until all abnormal discharge has ceased. To ensure that all follicular enlargements have been got rid of, a most careful scrutiny of the conjunctiva and all its folds has to be made. As an aid to such examination I have found the Binocular Magnifier, a modification of Berger's stereoscopic loupe, made for me by Messrs. E. W. Dixey and Son, of the greatest assistance.

To expose to view every part of the upper retrotarsal fold, after the eyelid has been everted in the usual way a second eversion has to be made. This second eversion is

not usually referred to in text-books, but is of the greatest aid both in diagnosing, and estimating the progress of, a case of trachoma. I find it can be invariably performed with the fingers unaided by the use of any instrument. The upper eyelid having first been everted in the usual manner, a little pressure backwards is made on the free border of the tarsus with the forefinger; this raises its attached border away from the globe so that the thumb can be easily inserted beneath it. With the forefinger on the everted free border and the thumb beneath the attached border, the tarsus can be lifted away from the globe, exposing to view the retrotarsal fold. To expose the extreme angles of the upper retrotarsal fold, it is often necessary to transfer the tarsus, whilst held in this manner away from the globe, from the forefinger and thumb of one hand to the forefinger and thumb of the other. I sometimes nip up the retrotarsal fold whilst it is held exposed between my two thumbs to express follicles from it with my thumb nails.

It is probable that when the conjunctiva has its adenoid layer replaced by fibrous tissue its powers of resisting the attack of micro-organisms becomes lowered. I have frequently seen cases with fibrous tissue formation in the conjunctiva of long standing become the subjects of fresh attacks of inflammation. An examination of the discharge has revealed the presence of Morax's diplobacillus and the use of sulphate of zinc has soon cured the inflammation. Such cases were clearly not recurrences of trachoma.

I do not think that the reappearance of a vascular condition of the cornea as an accompaniment of fresh inflammation in an old case of trachoma necessarily implies a recurrence of that disease. We know that the tracks of the blood-vessels in the cornea when pannus subsides never completely disappear. Slight causes will sometimes lead to their fresh distention with blood. I have known it to occur as the result of an injury and I believe it may follow on any

undue hyperæmia of the conjunctiva, such, *e.g.*, that produced by some form of intercurrent conjunctivitis.

Whether or not the presence of "Prowazek granules" or "trachoma bodies" is to be regarded as a diagnostic sign of trachoma has yet to be decided. Clausen,* writing on this point, says: "The individual granules cannot be recognised with certainty, or considered as forming a standard for the diagnosis of trachoma, but when the specific granules are present, whether the small or large ones, absolutely specific clusters of them can be found near the nuclei of the epithelial cells, or else lying free."

Most observers have failed to find any histological difference between the follicles in the conjunctiva in trachoma and in other affections. Mayou has pointed out as characteristic of follicles affected with trachoma the absence of plasma cells in them, the scarcity of cells undergoing mitosis, and the presence of central necrotic changes. So far, the differentiation of trachoma from other affections, characterised by the presence of follicular enlargements, has rested on a clinical basis.

The close historical resemblance of the follicles, when ever enlarged, has led some observers to regard all conditions in which such enlargements are met with as stages of one and the same disease. Most, however, are now prepared to agree that there is one affection characterised by follicular enlargements, which are liable to be followed by fibrous tissue formation in the palpebral conjunctiva and involvement of the cornea with pannus, and other affections accompanied by follicular enlargements which never under any circumstances have such sequelæ. In countries where trachoma is rife, and where the majority of the inhabitants suffer from it, the recognition of different classes of cases with follicular enlargement is very difficult. In countries or localities where the disease which has the serious sequelæ, viz., trachoma, does not occur, the occurrence of affections accompanied by follicular enlargements without such sequelæ is readily realised.

* Trans. XI Congresso Internazion. di Oftalmol., 1909, p. 854.

The conditions in which non-trachomatous follicular enlargements occur may be classified as follows :—

(a) *In Atropine and Eserine Irritation.*—In atropine and eserine irritation, a very wide-spread enlargement of the follicles in the conjunctiva is sometimes met with. The clinical appearances presented by such a condition has, I think, features of its own which are distinctive. At any rate, I have found the form, the appearance, and distribution of the follicles such that I have been able to diagnose the condition, when I did not know previously that the drug which caused it had been used. The follicles are always small, very numerous, always discrete, scattered over the retrotarsal folds and tarsal conjunctiva. They are translucent and superficial in these two particulars, differing from early trachoma follicles on the tarsal conjunctiva, which appear set deep in the tissue and look like grey opaque areas.

(b) *In Children with a General Tendency to Adenoid Formations.*—Careful examination of the retrotarsal folds reveals small follicular swellings in a very large number of children who make no complaint of their eyes, who have no abnormal discharge from them, and no undue redness. Such children are frequently found to be also the subjects of new formation of adenoid tissue in the pharynx and nasopharynx. In some children these follicular enlargements in the conjunctiva may be very numerous, especially in the lower retrotarsal fold. They form rows of little rounded protuberances, never bigger than a pin's head, and have no tendency to run into one another and become confluent. When large, their presence may give rise to some irritation, causing the child to rub its eyes, or giving rise to blepharospasm. In such cases, hyperæmia or inflammation may become superadded.

The diagnosis of this form of follicular enlargement from trachoma in its early stages rests on the position, size, and non-confluent character of the follicles. They are always located in the retrotarsal folds, never larger than a pin's

head, and do not run into one another. In the later stages of trachoma the tendency to fibrous tissue formation or involvement of the cornea are characteristic features.

(c) *In Mucopurulent Ophthalmia*.—As the result of the irritation of organisms which excite a mucopurulent ophthalmia, such as the Koch-Weeks bacillus, follicular enlargements may form in the retrotarsal folds. These remain discrete, and after a variable duration disappear, without either rupturing or causing any abnormal overgrowth of fibrous tissue.

To diagnose such cases from early cases of trachoma which commence as a mixed infection with acute symptoms, is often a matter of extreme difficulty. Indeed, in some it seems to me impossible to be quite sure of the diagnosis until the cases have been watched carefully for some weeks.

The presence of fibrous tissue formation in the conjunctiva, though such a frequent accompaniment of trachoma, is not pathognomonic of that affection. Besides occurring in the process of cicatrisation of injuries and burns, it may also be an accompaniment of other diseases.

In tubercle, in pemphigus, and in severe membranous ophthalmia portions of the conjunctiva become destroyed by ulceration. In the treating of these ulcers fibrous cicatricial tissue forms, producing an appearance closely resembling that seen in the late stages of a bad case of trachoma. So close may the resemblance be that the nature of the affection from which the condition arose can only be diagnosed by careful study of the history of the case.

Spring catarrh is another disease of the conjunctiva in which an extensive new fibrous tissue formation takes place. In this affection, unlike those first mentioned, the new formed fibrous tissue is not cicatricial fibrous tissue, it is not produced as a sequelæ of ulceration. It is one of the primary essential features of the disease. It grows forwards from the deeper layers, replaces the adenoid layer, and forms on the surface of the membrane the characteristic flat-topped elevations. In its non-cicatricial character it resembles

much of the fibrous tissue formation in trachoma, and the two diseases are also alike in the formation of large numbers of plasma cells which undergo degeneration, forming the hyaline material already referred to. Spring catarrh differs, however, from trachoma in the complete absence of follicular formation and also, as was first pointed out by Herbert, in the development of immense numbers of eosinophiles. A remarkable feature of the fibrous tissue formation in spring catarrh is the way in which it disappears as the disease subsides. It produces no contraction and leaves little if any sign of its former presence.

A CASE OF OBSTRUCTION OF THE CENTRAL VEIN AND GLAUCOMA; WITH REMARKS.

By Dr. TATSUJI INOUE (Tokyo).

(From the Pathological Laboratory.)

With Plates I and II.

OBSTRUCTION of the central vein with hæmorrhagic glaucoma is a disease which merits further study on account of its interest from the point of view of pathogenesis and pathological histology. The most recent papers dealing with the general aspects of the subject are those of Coats* and Harms,† who collected the published cases in which a pathological examination was carried out. Since then further additions have been made to the literature by Bartels,‡ Baquis,§ Coats,|| Verhoeff,¶ Tschirkowsky,** and Bauer.†† In the present paper a new case is reported.

A man, aged 66, was admitted to the R.L.O.H. on February 8th, 1908, under the care of Mr. Fisher, complaining of loss of vision in the left eye and deterioration of the right. He smoked 4 oz. of strong tobacco weekly, and drank a moderate amount of whisky. Urine acid, sp. gr. 1025, no albumen, no sugar.

In the right eye there was a small central scotoma for green; other colours recognised promptly. V. = 6/24, with +1.25 = 6/6. The sight of the left eye had been failing for three months at least. Cornea steamy, pupil inactive, tension +1.5, V. = perception of light in temporal field only. Eserin was ordered for both eyes.

* R.L.O.H. Rep., vol. xvi, p. 62, 1904.

† Arch. f. Ophth., vol. lxi, p. 1, 1905.

‡ Zeitschr. f. Augenh., vol. xiv, p. 103, 1905.

§ Beitr. zur Augenheilkunde, Festschrift f. Hirschberg. p. 33, 1905.

|| R.L.O.H. Rep., vol. xvi, p. 516, 1906.

¶ Arch. of Ophth., vol. xxxvi, p. 1, 1907.

** Klin. Monatsbl. f. Augenheilk., vol. xlvi, p. 272, 1908.

†† Arch. f. Augenheilk., vol. xliii, p. 13, 1909.

12.ii.1908. R. iridectomy.

26.ii.1908. R. quiet. No injection. Iris pattern blurred. Disc pale; no cupping. L. quiet, V. = bare perception of light. T. + 2.

4.iii.1908. R. quiet. T. n. Media not clear, disc not well seen, no marked cupping; V. with correction 6/12 and J. 2. L. T. + 1.

19.viii.1908. R. T. n. V. with correction 6/9. L. T. + 2.

3.ii.1909. Has not attended. No eserine used for three months. R. T. n. L. has been painful for one month, especially for the last 10 days. T. + 1.5. Much ciliary injection, cornea steamy. V. = no perception of light.

26.ii.1909. L. absolute glaucoma. T. + 3. L. excision, under chloroform.

28.iv.1909. R. no change. Field contracted to about 10° in nasal and lower part.

Pathological Examination (Plates I and II, Figs. 1 to 6).

The globe is fixed in Zenker's fluid, and divided horizontally.

The corneo-iridic angle seems to be occluded. The lens is *in situ*. The vitreous contains coagulum (probably chiefly due to Zenker fixation). The retina is sprinkled in all directions with hæmorrhages, which commence some distance out from the papilla, leaving the peripapillary zone free. The vessels are not visible at a distance from the disc; they do not seem to be very tortuous. The retina and choroid are *in situ*. The disc is slightly cupped.

Microscopical Examination.

The globe was embedded in celloidin, and sections were cut in a horizontal direction as far as the edge of the papilla. The nerve and nerve entrance were then excised, re-embedded, and cut in a frontal direction, so as to obtain transverse sections of the central vessels. About every fifth section was mounted. From these transverse sections the longitudinal section shown in Fig. 1 was plotted out according to the method of Harms. In this reconstruction all the transverse sections were not available; only those indicated on the scale were used—a number sufficient to obtain a general view of the changes.

Sections were stained with Ehrlich's hæmatoxylin and eosin, Heidenhain's hæmatoxylin, Weigert's elastic tissue stain,* Van Gieson's stain, and Birch-Hirschfeld's modification of Nissl's ganglion cell stain.

The *cornea* is normal; the endothelium well preserved. In the extreme periphery of the cornea, close under the epithelium, there is much vascularisation, representing the anterior edge of a considerable pericorneal congestion (Fig. 5). The vessels are filled with blood, and are surrounded by round cell infiltration. A few delicate vessels are found in the corresponding region of the periphery of the cornea.

Sclera.—The pericorneal and episcleral vessels are unduly numerous, and are filled with blood; the veins are very tortuous. The abnormal development of episcleral vessels extends as far back as the equator, where the vein walls are infiltrated with round cells in places, and show the features of a mesophlebitis; the lumen is not contracted, however, and the intima is intact.

The *corneo-iridic angle* is free from adhesion in three places on the temporal and one on the nasal side, representing in all about one-third of its whole circumference. Fig. 5 shows one of these areas in which the angle is free. These four non-adherent portions are separated by areas in which the angle is totally occluded. Where the angle is clear, the ligamentum pectinatum and its endothelium are partially preserved, but the adjacent stroma of the cornea and iris show much inflammatory infiltration and some cicatrisation, in the form of numerous round cells, polynuclears, lymphocytes, eosinophiles, pigment cells and clumps, and a layer of dense connective tissue. The endothelial lining of the angle is no longer regular or distinctly recognisable. The canal of the Schlemm contains blood in most of the sections, and its wall shows inflammatory infiltration.

The *anterior chamber* is of normal depth. Immediately in contact with the surface of the angle and the adjacent iris a

* According to Greeff (Guide to the Microsc. Examin. of the Eye, 1901, p. 79) and Bolles Lee (The Microtometist's Vade-mecum, edition 1900, p. 441) section should stain in Weigert's fluid for from 20 minutes to 1 hour. In my experience this is sufficient for paraffin, but not for celloidin sections; the latter should be placed in the incubator at 36° C., in a closed vessel, for from 3 to 24 hours. If this is done the section can be freely washed in alcohol without fear of decolorising them too much. (Fig. 4.)

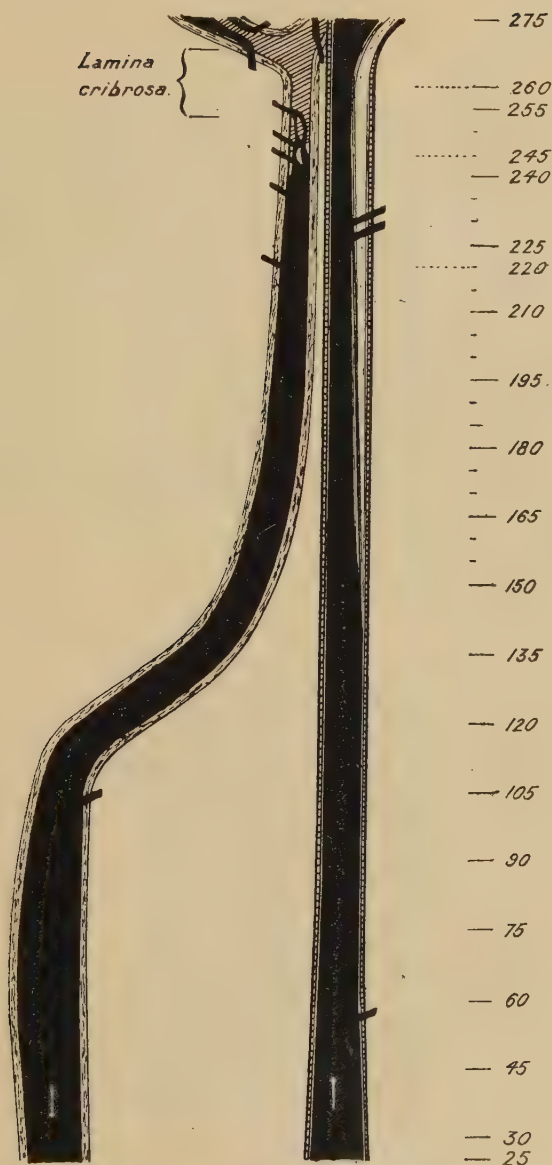


FIG. 1.— $\times 40$. The proliferation of the cells of the intima has caused a complete obstruction of the central vein in the region of the lamina cribrosa. The lumen of the artery is much contracted by proliferation of the subendothelial connective tissue. Thickness of the sections from Nos. 1 to 275 is $15\mu = 4.1\text{ mm}$. The number in the scale gives the number of the section in the series. Photographs are given of Section 260 in Fig. 2, of Section 245 in Fig. 3, and of Section 220 in Fig. 4.

PLATE I.

- FIG. 2 (Section No. 260 of Text-fig. 1, p. 27).— $\times 90$. The lumen of the central vein is completely obliterated by cells of the intima, with which many red corpuscles are mingled. The central artery is much contracted by proliferation of the subendothelial connective tissue.
- FIG. 3 (Section No. 245 of Text-fig. 1, p. 27).— $\times 90$. The lumen of the central vein is being re-formed by the accession of collaterals.
- FIG. 4 (Section No. 220 of Text-fig. 1, p. 27).— $\times 90$. Weigert's elastic tissue stain. The elastic membrane of both the artery and vein is normal. The vein lumen is re-established, and a small collateral is entering. The artery shows endarteritis, the lumen is displaced to one side, and between it and the elastic lamina there is some loose connective tissue (proliferated subendothelial tissue), which has shrunk away from the elastica, leaving an empty space (? artefact).

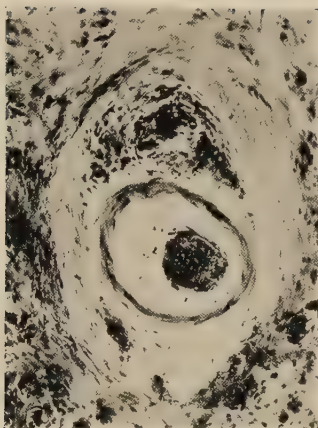


FIG. 2.

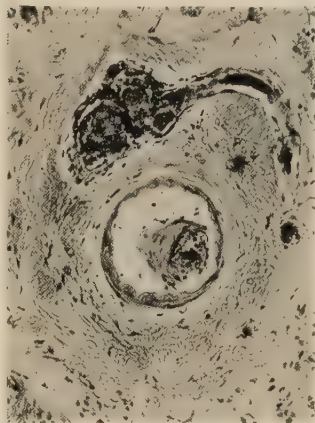


FIG. 3.

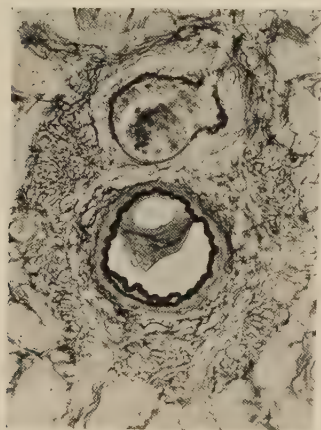


FIG. 4.

layer of fibrin is found in many of the sections (Fig. 5). Elsewhere in the anterior chamber there is a quantity of granular coagulated albuminous material, in somewhat greater amount than is normal; among it scanty round cells, and still more scanty red corpuscles, are found.

The *iris* measures 4 mm. in breadth, the pupil 2·5 mm. across. The iris stroma is more cellular than normal, and contains numbers of eosinophiles; the infiltration is greatest towards its anterior aspect. The surface is comparatively smooth owing to the presence of a layer of cells, mostly of connective tissue type, but partly round cells and pigment cells; it is lined anteriorly by the endothelium. The posterior surface of the iris is intact. The sphincter is somewhat atrophic.

The *ciliary body* is normal in size and not infiltrated. The epithelium is proliferated in places, otherwise intact. A few round cells are present between the layer of the epithelium and on its inner surface; there are no blood corpuscles in these situations. The vessels and ciliary muscle are intact.

The *lens* is normal; capsule intact.

As a whole the *choroid* is normal. Anteriorly the stroma is atrophic, especially on the temporal side, where it is much thinned, and the vessels are invisible in certain areas. The *venæ verticosæ* are normal. The long and short ciliary arteries are practically normal.

The *vitreous* fibrils are normal, but seem to contain more than the usual quantity of albumen. Granular albuminous coagula are not found. A very few scattered round cells and blood corpuscles are found, especially on the *limitans interna*.

In the *retina* the rod and cone and outer nuclear layers are on the whole normal. The inner nuclear layer is atrophic. The ganglion cells are very scanty and contain no Nissl granules. In the macula the tissue seems to be oedematous and the nerve fibres and ganglion cells are especially atrophic. There are no hæmorrhages in the macula. Many small hæmorrhages are present near the vessels, especially in the neighbourhood of the papilla; in the immediate vicinity of the disc, however, where the larger vessels run, there are relatively few hæmorrhages, and none are found within some little distance of the *ora serrata*. The hæmorrhage is mostly in the nerve fibre, ganglion

PLATE II.

FIG. 5.— $\times 60$. At this point the angle of the corneo-iridic angle is open. The anterior chamber contains a layer of fibrin, immediately in contact with the surface of the angle; elsewhere there is a granular coagulated albuminous material. The tissues adjacent to the angle are infiltrated. The canal of Schlemm contains blood. Considerable pericorneal congestion and round cell infiltration.

FIG. 6.— $\times 90$. A large hæmorrhage in the retina. In the centre the blood corpuscles are broken down, and take on the hæmatoxylin stain more than usual.



FIG. 5.

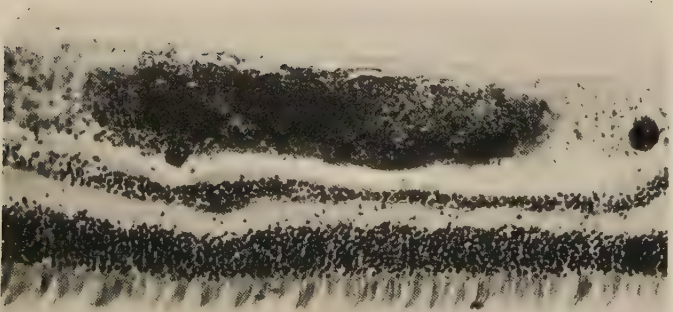


FIG. 6.

cell, and inner reticular layers, frequently reaches the inner nuclear, and extends exceptionally as far as the outer nuclear layer. The blood corpuscles are mostly well preserved, but are broken down in the centre of a large hæmorrhage (Fig. 6), and in that situation take on the hæmatoxylin stain more than usual. The large arteries show, in general, considerable thickening of their walls, and have undergone hyalin degeneration. Calcification is present in the wall of a medium sized artery. The vein walls are also thickened, but not so much as in the case of the arteries. In a vein above the papilla the lumen is completely occluded by endothelial proliferation, similar to that which is present in the central vein.

Optic Nerve.—The papilla shows only slight excavation. The nerve fibres are much atrophied, while the neuroglia and the fibrous tissue are proliferated.

The *Central Artery* has a normal membrana elastica, muscularis and adventitia. At the level of the lamina cribrosa and for some distance above it the lumen is much contracted by proliferation of the subendothelial connective tissue. A simple regular layer of endothelium lines the lumen. The proliferation of the subendothelial tissue, *i.e.* the tissue between the endothelium and the elastic membrane, is much greater on one side than on the other, where the endothelium lies directly upon the membrana elastica. On the side where proliferation is greatest, fairly compact connective tissue is present under the endothelium; it has a triangular form in the sections. Between the compact fibrous tissue and the muscular coat, loose connective tissue cells are present, which in places are quite isolated from one another. The compact fibrous tissue is coloured red with Van Gieson's stain, and with the elastic tissue stain shows some transversely arranged elastic fibres (Fig. 4). Posteriorly the lumen gradually expands till it again becomes normal (Fig. 1).

Central Vein.—In front of the lamina (Fig. 1, Section 275) two veins are seen in the section, of which the lumina are contracted by proliferation of the intima in such a manner that each contains two small channels. At the level of the lamina cribrosa (Fig. 1, Section 260; Fig. 2) the lumen is completely obliterated by proliferated cells of the intima; many red corpuscles are mingled with these cells, but do not form a

distinctly demonstrable blood channel. About half the red corpuscles have become confluent. The blood corpuscles and cells of the intima are so mingled together that a blood current through the tissue must have been very difficult, but one cannot altogether exclude a very slow stream. That these cells of the intima are all endothelial in origin is very improbable; perhaps other cells are mixed with them, such as connective tissue cells, round cells, and other cells contributed from the blood stream and from the subendothelial tissue. The adventitia takes no part in the narrowing of the lumen, while the elastic membrane occupies nearly its usual position, as can be shown by elastic tissue staining. In a transverse section of the nerve behind the lamina (Fig. 4) the proliferation of the intima is visible, while the elastica preserves its normal relations. Small collaterals run into the vein above the obstruction (Figs. 3 and 4), and are especially numerous close to the posterior surface of the lamina. In consequence of the accession of the collaterals the vein lumen gradually becomes larger, and finally reaches normal dimensions. Hæmorrhage is present in the sheath of the vessels, at the lamina cribrosa, where the obstruction is present, but not further back.

Summary.

In this case it is not absolutely certain from the history whether the obstruction of the vein or the glaucoma was the primary disease. In view of the figures to be given presently, however, it is in the highest degree probable that the obstruction preceded the glaucoma.

The chief interest of the case lies in the state of the corneo-iridic angle, which was only partially occluded. The obstruction of the central vein was probably due to proliferation of the endothelial and subendothelial tissues.

OBSTRUCTION OF THE CENTRAL VEIN AND GLAUCOMA.

It is an important question whether the glaucoma precedes or follows the obstruction of the vein. As material for the solution of this problem, I have used all the cases in

the literature since 1892, in which complete or almost complete obstruction of the central vein was proved by pathological examination, and in which the dates of the occurrence of the obstruction and of the glaucoma were given with some exactitude. These are the cases of Wagenmann, Weinbaum, Purtscher, Alt, Bankwitz, Würdemann, Coats, Sidler-Huguenin, Harms, Baquis, Verhoeff, Tschirkowsky, Bauer; in all 32 cases.

Among these, obstruction was first and glaucoma followed in 31 cases; in one case only, that of Bauer,* a chronic simple glaucoma preceded the venous obstruction. In these 31 cases the shortest period between the obstruction and the glaucoma was in one of Coats's cases,† in which the interval was only 13 days; the urine contained abundant albumen. The longest interval is at most 11 months; in one of Harms's cases‡ it was "less than 11 months." In many other cases the exact dates are difficult to decide, because the patient frequently did not consult the surgeon at the beginning of the first attack of glaucoma. If, therefore, the calculation were made from the time of the loss of vision to the time of the patient's first visit (these being often the only data available in the clinical report), too long an interval would be obtained between the onset of obstruction and the onset of glaucoma. The figures will give a maximum number of days beyond which the interval could not have extended, though it might have been less.

This average maximum number of days was found to be 105 in the 31 cases; allowing for the error above mentioned, the usual average interval between the obstruction of the vein and the subsequent glaucoma may therefore be put at about 100 days, or rather less.

These statistical considerations are important in inquiring whether or not a causal connection exists between the two diseases. According to Wagenmann§ the two phenomena,

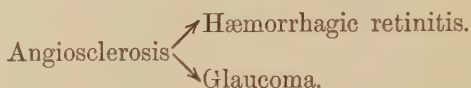
* *Loc. cit.*, Case 1, p. 16.

† *Loc. cit.*, Case 1, p. 75.

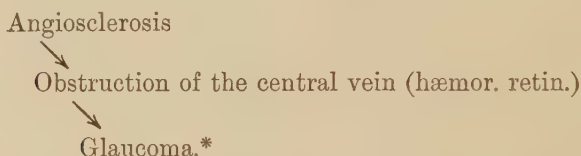
‡ *Loc. cit.*, Case 8, p. 135.

§ *Arch. f. Ophth.*, vol. xxxviii, p. 258, 1892.

hæmorrhagic retinitis and glaucoma, are really independent of one another, but are both dependent on a common cause, viz., angiosclerosis; this explanation may be expressed as follows:—



I would admit this for only a very small proportion of the cases. The statistics given above show that in most instances the formula should stand thus:—



If this schema be correct there still remains the problem: in what manner obstruction of the vein causes glaucoma. In looking over the literature of the subject, for instance, in the paper of Coats† or Harms,‡ it is evident that the pathogenesis is enveloped in uncertainty. An hypothesis is wanted to explain how glaucoma is brought about by obstruction, a question of considerable importance in obtaining clear views on these complicated processes. In my opinion the following is the sequence of events.

The obstruction causes retinal hæmorrhage; the blood corpuscles in disintegrating produce toxines; the toxines are continually carried to the angle of the anterior chamber, owing to defective drainage by the central vein and its collaterals; the tissues about the angle become inflamed, an adhesion is formed, and glaucoma is the result.

In the earliest stages there may be no adhesion, but only inflammation at the corneo-iridic angle. This was shown

* From another standpoint Coats has deduced the same relationship (*loc. cit.*, p. 111).

† *Loc. cit.*, pp. 112 and 555.

‡ *Loc. cit.*, p. 149.

in a case of Verhoeff's,* in which the eye was excised six days after the commencement of glaucoma.

When, for instance, a hæmorrhage occurs in the brain, the corpuscles disintegrate, and albuminous substances are produced which exercise an attractive influence on the adjacent vessels (hæmotactic action). These blood-vessels proliferate and cicatricial tissue is formed at the site of the hæmorrhage. In the case of retinal hæmorrhage also the disintegrating corpuscles† produce hæmotactic albuminous substances (toxines). These albuminous substances will show their effects where they most accumulate, namely, at the angle of the anterior chamber. It seems to me that the condition of glaucoma after obstruction of the vein is only what one might expect. The hæmotactic nature of the toxine must be assumed, since otherwise it is difficult to understand why hæmorrhage should so easily occur not only from the retina, but also, at operations, from the corneo-iridic angle and the iris; why the canal of Schlemm so frequently contains blood in the sections; in short, why there is so great a tendency to hæmorrhage from almost all the tissues of the eye.

The objection might be raised: "Why does not glaucoma follow in all cases of thrombosis?" It appears to me that this is analogous to the question: "Why does not tobacco amblyopia follow in all heavy smokers?" In the case of tobacco amblyopia it is well known that lowered nutrition of the patient and the simultaneous abuse of alcohol have a notable influence. In the case of hæmorrhagic glaucoma after obstruction of the vein an important secondary part may be played by angiosclerosis, albuminuria, etc. Tobacco amblyopia is, of course, only mentioned as an illustrative analogous instance, not as a proof of the explanation here advanced.

Although the evidence in support of this toxine theory

* *Loc. cit.*, Case 1, p. 3.

† It is certain that the red corpuscles in the retina disintegrate (see Fig. 6).

is in certain points insufficient, and although it is not improbable that the mode of stating it may be in some respect faulty, yet I believe that along these lines further light may be sought on the obscure subject of hæmorrhagic glaucoma.

In conclusion, my thanks are due to Mr. Coats for his friendly help and advice in the course of this work.

RETINITIS CIRCINATA, AND ITS RELATION TO OTHER
FORMS OF RETINITIS WITH HÆMORRHAGES AND
EXUDATES.

By J. HERBERT FISHER.

HAVING recently read in vol. xvii, part iii, of the Roy. Lond. Ophth. Hospital Reports the paper by Coats entitled "Forms of Retinal Disease with Massive Exudation," I was led to review my clinical notes of a case of this description which I had under observation some years ago. The ophthalmoscopic appearances in one eye of this patient are characteristically identical with Coats' cases, while those of the other eye suggest so strongly retinitis circinata in addition to massive exudation in the retina, that the idea of a pathological similarity between the two conditions can hardly be resisted. I therefore propose here to report my case, and at the same time to collate with it certain other cases allied to circinate retinitis, which have come under my observation. I regret that I am unable to supply ophthalmoscopic drawings and that I must rely on the descriptive power of words to bring the cases before the mind. I confess also my own deficiencies in pathology, but the consideration of my cases will, I hope, prove suggestive as to future investigations by those competent to conduct them.

The cases reported by Coats need to be carefully studied in the original; they are six in number. In nearly all of them the massive exudation in the retina was peripherally situated or in any case most marked in the periphery. In the cases in which an ophthalmoscopic examination had been made, some vitreous opacities were frequently present; the mass itself caused a prominent boss and was usually of a greyish white or yellowish white colour; and hæmorrhages were in all cases associated with it, and frequently lustrous crystalline spots, regarded as cholesterin. Pathological

examination of the specimens led Coats to the conclusion that the masses were primarily of hæmorrhagic origin, and that the pouring out of blood took place first in the outer reticular layer of the retina, the outermost layer to which capillaries from the retinal vessels reached, and then further extended to form a localised collection in the sub-retinal space. The added tissue, at the date when the specimens were examined, in most cases consisted of fibrous tissue in various stages of development or degeneration; red blood corpuscles were recognisable, and cholesterin crystals were regarded as corroborative of the hæmorrhagic origin of the mass—calcareous and even osseous change was observed in some of the cases, and giant cells and ghost cells were constantly found in association with the exudates. The inner layers of the retina were as perfect as could be anticipated under the conditions, while the layers including and external to the outer reticular layer were totally disorganised and destroyed in the diseased area. The choroid was intact and very little if at all altered, while the lamina vitrea was uninterrupted and frequently showed traces of the pigment epithelium, as it bounded the retinal masses on their external aspect.

Coats took his own six cases and, collecting other reported cases from the literature, classified them under three headings, viz.: 1. Those without gross vascular disease; 2. Those with gross vascular disease; 3. A peculiar group, perhaps allied to the previous two, which is characterised by the formation of large arterio-venous communications.

With two of his own cases Coats placed about 23 others in group 1; this class was found almost always in young people; the average age was less than 20 years; the disease preponderated in males, and was bilateral in only two instances—in some cases the changes were peripheral, in others central; hæmorrhage was attributed as the primary initial change by some observers; straining in whooping cough was suggested as a determining cause for the bleeding in one case, by Nettleship; a history of epistaxis was

elicited in two instances; the onset of the changes was frequently insidious and the progress of the disease a very slow one, extending over many years. Often there was a detachment in some part of the fundus, but over such an area the retina was supported and not freely floating. Associated with the mass of white exudate were usually hæmorrhages and cholesterin spangles. The diseased process appears to become quiescent.

Coats, in group 2, placed four of his own cases and seven from literature. Apart from the vascular disease this group closely resembles group 1. The average age was 21 years, males preponderate; there was no record of a bilateral case. The mass was more often in a temporal situation than elsewhere—white spots were frequently present in other parts of the fundus, and the exudation had a certain tendency to follow the retinal vessels. The smaller vessels, both arteries and veins, in this group showed extraordinary forms of vascular disease; the vessels were often fusiform or beaded; minute expansions occurred, on or by the side of the vessels, and the arteries sometimes ended in small knobs. Loops, kinks, spiral tortuosities, brushes and glomeruli, formed of vessels previously invisible, and new formed anastomoses, are described. The more common forms of vascular disease such as enlargement, tortuosity, and white sheathing were also present. Coats has been unable to satisfy himself whether the changes in the vessel walls are primary, or are the result of the retinal hæmorrhage and the subsequent changes which the blood mass undergoes. Groups 1 and 2 are in all other respects practically identical.

Group 3 appears to belong to a different class. Young patients, again, furnished the examples of this group; the enormous size of arteries and veins seems conclusive of arterio-venous communications; the vessels end in large cystic swellings of a reddish or reddish-yellow colour, which pathological examination has proved, in the three cases which alone have been so examined, to be a new growth of a vascular tissue similar to a capillary angioma.

In groups 1 and 2 Coats regards hæmorrhage as the primary lesion upon which the subsequent changes depend, except that in a few cases vascular disease may be the condition antecedent to the hæmorrhage; other causes for hæmorrhage may be stress at birth, alterations in coagulability of the blood, and strain or injury.

It will be convenient at this point briefly to recapitulate what is known of the pathology of the well-defined disease known as retinitis circinata. The well known girdle of oval or reniform spots needs no description; the retinal vessels are in all cases superficial to them; the spots occasionally coalesce into larger plaques, and may, rarely, be appreciably raised; the fovea is greyish or slightly opaque: detachment has been noted here. A minimum of pigment disturbance is associated with the affected area; the disease occurs in the later years of life; the retinal vessels are often tortuous or sclerosed, and hæmorrhages are the rule; cholesterin crystals are often present. As to the nature of the spots, Fuchs regarded them as coagula of albuminous fluid, and he and Nuel denied that they are degenerated blood clots; de Wecker considered them due to the fatty degeneration of extravasated blood. Amman has made the only pathological examination which has been reported. The patient had albuminuria. Amman found hyaline masses in the outer reticular layer, and some of these showed that they were undoubtedly derived from red corpuscles, all the different stages being traceable. Large mononuclear bladder-like cells, filled with fat globules (?ghost cells) were found; the retina was about four times its normal thickness in the situation of the spots, the thickening being chiefly in the outer reticular layer; in the region of the exudates the rods and cones were absent or destroyed. Parsons considers the evidence insufficient to enable us to be positive that all the changes are dependent on previous hæmorrhage, and thinks that albuminous coagula allied to those found in renal retinitis may play a part—he regards the condition as essentially degenerative and not inflammatory. Fuchs

has observed the spots and hæmorrhages to slowly disappear in a patient whom he kept under observation for four years.

It is unnecessary here to describe the typical case of retinitis circinata. A characteristic case is described and illustrated by Spicer in vol. xiv, Trans. Ophth. Soc.; in vol. xvi other cases are described, and some are accompanied by ophthalmoscopic drawings, by Lawford and Laws, and others, doubtfully belonging to this class, by Hartridge and Doyne; in vol. xviii, E. C. Fischer reported and figured another example. In some of the cases hæmorrhages accompanied the retinal wreath: in Spicer's and Lawford's cases central hæmorrhages were present in the opposite eye. In Fischer's case the ring was sufficiently thick to cause an appreciable elevation of the fundus level. I reported a case briefly in vol. xxii, Trans. Ophth. Soc., which I had the opportunity of watching for over 12 months: the retinal vessels were the subject of some sclerosis; no hæmorrhages were ever detected; the ring of retinal change became more complete during the year that the patient remained under my observation.

The following case has not been reported—it appears to me to support the view that the white spots of circinate retinitis are secondary to blood extravasations in the deeper layers of the retina, and it confirms Fuchs' statement that the exudates may disappear.

J. W., male, æt. 62, was seen by me at Moorfields Eye Hospital in April, 1902. He was a commercial traveller, and appeared to have indulged in alcohol. He complained that his eyes were watery, and that his sight had been getting misty for three or four months. V., R. = fingers. L. 6/24; $\bar{c} + 2.50$ sph. = 6/12. The urine was normal. The right fundus showed the typical elliptical wreath of white dots surrounding the macula in the manner characteristic of retinitis circinata; at the macula was the usual greyish mottled appearance; temporally situated in regard to this first ring, but fusing with the upper outer part of its circumference, was a second circinate ring; this second ring

was somewhat smaller than the primary one, but composed of exactly similar dots ; there was no retinal mottling in the centre of the secondary ring ; the dots of each ring were deep to the retinal vessels ; running as a tangent to the upper circumference of the secondary ring was a rectangular ribbon of white change or exudate, not composed of spots but evenly diffused ; it was about $\frac{1}{2}$ P. d. in width and 3 P. d. in length ; it was dead white in colour, and may have been an œdema of great density. Mr. Gunn, to whom I showed it, thought it likely it was of this character. This ribbon-shaped band lay in its whole length behind a large retinal vessel. The retinal vessels appeared healthy.

In the left eye an arch of evenly diffused retinal hæmorrhage extended out above the macula from the upper outer part of the optic disc margin ; it lay in the concavity of the superior temporal vessels ; it terminated well beyond the macula in some irregular splashes of blood ; there was no hæmorrhage below a horizontal line drawn through the centre of the disc. A week later I observed a retinal hæmorrhage in the right eye ; it was situated just within the secondary circinate ring adjacent to the part where the two rings fused ; at this date, in the left eye, the band of blood was still present, of the same arching shape, but was becoming interrupted as if breaking up into segments ; outlying splashes of blood continued the outer end of the arch lower down, and one patch of blood was now present almost directly below the macula.

The case was repeatedly examined : the white ribbon-shaped band described at first as running tangential to the upper circumference of the secondary ring in the right eye seemed in the course of a month or so to fuse with the dots composing the adjacent part of this ring, and to turn down at its outer end and become less homogeneous as it did so ; the blood arch in the left retina gradually became more broken up into patches ; the patches became first darker, then in the concavity of the arch a yellowish hue appeared, while by the end of May the arch presented a whitish speckled appearance at its temporal extremity. In the right eye hæmorrhages were noted in relation to the dots composing the secondary circinate ring from time to time, and the secondary ring became even more perfect : the hæmorrhages

were noted in relation to the secondary ring, none in relation to the, presumably older, primary ring; *e.g.*, June 11th, 1902: "hæmorrhagic spots on the white exudate of the lower part of the secondary ring, just where it is about to join the primary ring"; July 9th, 1902: "fresh hæmorrhage just within the outermost part of the circumference of the secondary ring." On July 29th, 1902, Dr. James Taylor thought the patient "probably the subject of mitral stenosis; some irregularity of heart and a feeble beat." Digitalis and nux vomica were given.

On November 19th, 1902, I noted:—"The changes in the secondary circinate ring of the right eye are certainly more extensive; there are also some hæmorrhages on its outer part." The appearances in the left eye had greatly altered. A faint bloom adjoining the upper part of the disc was all that remained to indicate the presence of the previous arc of hæmorrhage, and this bloom only reached a short distance from the disc edge. A large white plaque rather larger than the disc, and of roughly quadrilateral shape, lay just above the disc, and was separated from it by a narrow band of normal fundus; there was a large dark hæmorrhage upon the plaque; a little recent red blood was observed in the retina to the outer side of the plaque and above the ascending temporal vessels. I learnt that the patient suffered from palpitation and shortness of breath, and had to sit up in bed at night; his habit was to rise at 5 a.m., and he was very active all day. I ordered him to be less active, to spend 12 out of every 24 hours in bed, and to lie down for two hours every afternoon. This advice he was able to follow, and in March, 1903, reported that he had much improved in general health as the result. Ophthalmoscopically I observed that the primary circinate ring in the right eye had broken up a great deal, and was less typically circinate; while the large white plaque above the disc in the left eye was elongating into a ribbon-like band.

I saw the patient again, 13 months later. I noted: "There is no remnant of anything like circinate retinitis in either eye now. Much old exudate in deeper retinal layers, with pigment scattered over its surface in central part of the fundus of right eye; changes in the left fundus were of similar character to those in the right, but less extensive; a sharply-cut organised exudate was still seen to run a short distance up and out from the disc

of the left eye. No hæmorrhages were found in either." The best vision of the left eye was now 6/24. The fields were perimetrically charted, and found to be practically full; a scotoma was recognisable in the upper nasal portion of the right field.

The points that I should like to emphasise at this stage are, that the above case supports the view that hæmorrhage forms the basis upon which are formed the spots of which the exudate in retinitis circinata is composed; that the circinate rings may disappear; in Coats' cases the hæmorrhage takes place at first into the outer reticular layer, and, as it increases, invades the subretinal space; that in retinitis circinata the exudates were found by Amman to lie in the same outer reticular layer; Coats considers that the leucocytes which succeed in organising the blood clot are derived from the retinal vessels; presumably it must be by the energies of similar leucocytes that such radical changes as took place in the more limited exudates of the case above described are effected. May it be that the ribbon-shaped white band I noted adjacent to the upper margin of the secondary circinate ring in the above case was due to the army of leucocytes as they passed from the retinal vessels which overlay the middle of this striking feature of the ophthalmoscopic picture? With such a diapedesis would be an œdema; the band appeared to fuse with the circinate dots to which it was contiguous; the circinate rings in time totally vanished.

I should like now briefly to draw attention to a case of degeneration of retinal vessels associated with cardiac disease in a young boy. In association with the local arterial degeneration there developed an incomplete ring of dead white dots corresponding in appearance, situation, and depth in the retina with those of retinitis circinata.

The case is described by me in vol. xxiii, Trans. Ophth. Soc., p. 73, illustrated by a black and white reproduction of a coloured ophthalmoscopic drawing.

The patient was a boy, 13 years old, who showed no signs of inherited syphilis, though his mother had experienced with her family a large infantile mortality.

Rheumatic fever, when he was 5 years of age, had left an incompetent mitral valve, and the heart was large and dilated, with feeble sounds.

The retinal exudate was in the right eye, and formed the upper outer segment of what would have been a typical circinate ring if it had been complete. There was a slight mottled disturbance at the macula. The dots of retinal exudate were on the whole smaller than in most cases of ordinary circinate retinitis; they lay deep to the retinal vessels; similar spots tended to follow the course of some of the retinal vessels, as the latter branched off from the superior temporal trunks. Up and out beyond the main incomplete arch of retinal dots some secondary branches of artery showed an extreme silver wire appearance, and on three small twigs multiple minute aneurismal dilatations were easily recognised; a few small spots of hæmorrhage were observed near the diseased arteries. The peripheral veins of the fundus were inclined to be beaded. In the temporal and upper nasal periphery of the fundus were found a few other spots of similar white exudate in the retina, and in the latter situation were a few other examples of local arterial degeneration and dilatation. The left fundus was in every way normal.

The case, I regret to say, was under observation only for a period of about two months, and I am unable to give the sequel. Probably the patient did not live long; the heart was in an extremely bad state.

I have introduced the case here as it seems to suggest that, if the exudates of circinate retinitis are of hæmorrhagic origin and in so far are related to Coats' cases, differing in the fact that the hæmorrhage does not invade the sub-retinal space, and if the ordinary case of retinitis circinata is due to vascular degeneration, given the unusual condition of vascular degeneration in early life changes of a circinate type may occur in childhood.

Coats suggests that vascular degeneration may be one

but by no means the only cause of the hæmorrhage in his cases which occur in young patients; those who are satisfied that retinal hæmorrhage is the first stage towards the formation of a circinate ring will probably be ready to admit that vascular degeneration, usually senile, is the necessary postulant to the hypothesis.

A. G. L., male, æt. 22, was seen by me at Moorfields Eye Hospital in October, 1902. He is another example of retinitis of circinate type in a young patient who was the subject of uniocular arterial disease. The vision of the left eye had been worse than that of the right for years, but had been especially failing during the preceding six months. Its vision was now 6/36. The right eye was in all respects normal. The left fundus showed a ring of retinal exudation of great brilliance surrounding the macula; it was of elliptical shape and quite like retinitis circinata; the wreath was deficient below; there were a few small hæmorrhages in the circumference of the ellipse down and out. The arteries of the fundus were of the silver wire type. My assistant reported a doubtful trace of albumen in the urine; I examined a specimen passed the first thing in the morning a week later, and found neither albumen nor sugar. At his second visit I noted there was no mottling at the macula itself; with dilated pupil I found in the inner and upper periphery much pigmented old choroido-retinal exudate or scarring; white sheathing of veins; one vein ran upwards, and just before reaching the peripheral change above alluded to suddenly developed thick white lines for about 1 P. d. in length; here the blood stream became greatly reduced; at intervals, while I was observing this point, the blood current would diminish to a thin red line and then expand again; I could not appreciate that respiratory movements had any influence over this rhythm. In the outer and lower parts of the fundus periphery I found two or three areas of circular shape where recent exudate in retina or choroid seemed to be present; there were also one or two larger patches of older exudate of brilliantly reflecting nature.

The patient's appearance and the history in no way pointed to syphilis, but my note at the time was: "The suggestion is

syphilitic choroido-retinitis, syphilitic arteritis, and the circinate retinitis secondary to arterial disease." This opinion is open, of course, to discussion. The case seems sufficiently interesting to introduce here, and especially so in reference to Coats' group 2 cases, for on October 25th, 1902, I noted "condition as before, but up and out some gleaming reflecting spots, which, when light falls obliquely upon them, reflect it in lines and streaks ; in the same district of the fundus are two retinal hæmorrhages on a greyish spot, and close to these a small coil of vessels very like a cirroid aneurism ; this clump is distinctly elevated."

I now come to my last case, which seems to me to afford a very strong connecting link between Coats' cases and retinitis circinata.

The patient was Miss S., æt. 29, whom I saw privately in October, 1898. The family is well known to me, and there is no suggestion of inherited syphilis. Two maternal aunts had died of phthisis, and her maternal grandparents both died of chest trouble, which in one case was tuberculous. On her father's side the family history was good, and both Miss S.'s parents are at the present date alive and well. Miss S. had had a good deal of worry ; had been anæmic for years, though living in the best of seaside air ; the bowels were inclined to be constipated. The catamenia had never been satisfactory ; for years the periods had been in abeyance ; subsequent to that very slight and irregular, but for a few months before I saw her had been more regular but scanty. Her nose had bled slightly on one recent occasion. Miss S. thought she had had rheumatism ; her mother had had rheumatic fever and now suffers from osteo-arthritis.

The right eye had been regarded as defective for years and tended to diverge, but no exact information could be given in regard to it. A week before Miss S. saw me she became alarmed at noticing a black spot in the direct sight of the left, her good eye ; it seemed to have got more dense for 24 hours and then to have remained stationary. For many years she had noticed phosphenes and also micropsia with this eye, *e.g.*, on rising from a chair or on blinking the eye.

Miss S. saw 5/6 with her R. eye and some letters of 5/5 with a convex lens.

Her L. eye saw 5/36 with difficulty, and best by excentric fixation. Looking in my face with her left eye she saw it dull and black; my hands held in the periphery of the visual field were much brighter; the one held in the temporal periphery was more distinct than that held in the nasal periphery.

Ophthalmoscopically I noted in the right eye a slight, fine, vitreous haze; in the extreme temporal periphery a white glistening globular area; the retinal vessels were in front of it, and were raised up by it till they were in focus with a +7 O.D. or a +8 O.D. lens; there was a little pigment change near the central limit of the mass; it all appeared of old date and inactive; there were no changes of any sort in the central part of the fundus.

In the left retina I found a large horseshoe shaped patch of white exudation with slight retinal pigment disturbance; the heels of the shoe embraced the disc above and below, and were composed of discrete small white spots; at the toe of the shoe, which lay to the outer side of the macula, the spots had become confluent, and formed large glistening plaques; there was a little pigment change adjoining them; the shoe therefore surrounded the macula, and the macula itself appeared grey and œdematous. From the heels of the shoe similar dots, small round or oval, extended far towards the upper and to a less extent towards the lower periphery; the spots here were abundantly scattered along the lines of ascending and descending retinal vessels, and the vessels lay anterior to them. The toe of the shoe was of a width equal to 2 P. d.; beyond its convexity towards the retinal periphery the retina was cloudy, œdematous, and swollen about 4 D., while in three or four places adjacent to vessels small oval spots of faint hæmorrhage were recognised; the œdematous state of the retina extended to my extreme ophthalmoscopic limit.

She was sent to a physician: he found the urine normal, detected nothing but some anæmia and a cardiac murmur which he regarded as hæmic; a blood count and examination by a skilled pathologist revealed nothing more. No enlargement of lymph glands or spleen, and all organs sound.

A further ophthalmoscopic examination a few days later showed no material change, but I found the mass of exudate and tissue at the temporal periphery of the right eye was even more prominent than I had on the first occasion estimated it to be. The conclusion I had come to was that the whole condition of the retina was circulatory and hæmorrhagic in origin, and dependent upon the blood state.

On October 31st, 1898, she was seen at my request by Mr. Nettleship in conjunction with me. He confirmed my observations and deductions in all particulars. He found the temporal swelling in the right fundus as high as 10. D.; drew attention to one small local swelling on a vein in the left fundus; and ascertained that she suffered from chilblains and was prone to local syncope of one finger; he pointed out one vein over the right swelling which became white and thread-like.

She was treated by total rest of eyes, and dark goggles, took arsenic and cod liver oil, and was generally instructed by the physician to whom I had referred her.

On November 9th, 1898, there was less œdema in the left retina; spots of hæmorrhage and general appearances, except for slightly more definition, were as before.

On April 26th, 1899, the vision of the left eye had improved to 5/9 partly. She thought the black disc had passed somewhat to one side. Ophthalmoscopically the main changes were the same, but œdema in the concavity of the horseshoe had quite subsided; there were two large areas of yellowish exudate flecked with blood in the lower outer part of the retina, one above the other.

On May 5th, 1899, she saw Mr. Nettleship again: he found vision of left 6/6 partly. Mr. Nettleship drew attention to a small elliptical red swelling with a white reflecting centre situated on an artery in the temporal periphery of the right eye—he regarded the spot as an aneurismal dilatation. I was not satisfied that it was of this character.

On November 5th, 1899, the direct vision of each eye was practically full. The patient thought the black disc had moved still more out of line of the direct vision of the left eye. She still experienced symptoms of photopsia occasionally. I noted that the fundus changes in the right were quite unaltered.

I could not confirm the recognition of any aneurism ; though the arteries were strikingly bright they did not obstruct veins at points of crossing. In the left eye the changes were of the same nature as before but evidently more quiescent ; no œdema, and no hæmorrhages now present ; the macula show a somewhat spotted and speckled appearance. I noted two or three gleaming spots of cholesterin crystals associated with the toe of the horse-shoe exudate near its upper part. In the outer part of the fundus beyond the toe of the horseshoe I found several circular reddish spots with white centres similar to that which Mr. Nettleship had regarded as an aneurism in the right eye : two of these lay close to an artery, there were others less striking ; I was unable to be sure of their nature or significance ; the general health was by now much improved ; occasionally slight nose bleeding occurred.

In October, 1900, vision full, as before, with either eye. In the left eye the exudate had become more of a plaque, *i.e.* discrete dots appeared to have been fusing together ; an extension of the lower limb had carried it to a termination now found to lie slightly down and in from the optic disc ; the temporal part of the horseshoe was now represented by a dead white exudation plaque crossed by retinal vessels ; at the extreme temporal periphery exudation had collected into a small globular swelling raising up the retina to a height of several diopeters over it ; the retina here did not float. I saw no hæmorrhages. The patient was in good health after a holiday in Switzerland. She stated that after exercise her vision was generally temporarily worse, and seemed to clear on her resting quietly ; this phenomenon was not due to any vitreous opacities, and I think indicated the circulatory nature of the trouble.

In November, 1901, I fancied the upper limb of the horse-shoe rather less perfect : the only other development was the presence in the outer periphery of the left eye, beyond all the exudation, of what appeared like a small cystic detachment of the retina which I thought had fluid behind it. In November, 1902, the conditions were much the same : I observed one hæmorrhage at this date in relation to exudation. I have not seen the patient since, but have no doubt I should have done so had any material change in subjective symptoms occurred.

In Miss S., then, I present a case which, in the right eye, was discovered to show changes evidently of long standing and quiescent, quite identical with those of Coats. In her left eye was watched over a long period the process of development of similar massive retinal and subretinal exudates, and associated with them was a type of central retinal degeneration of the closest resemblance to retinitis circinata. The patient was young, suffered from anæmia with menstrual disturbance, was inclined to epistaxis and suffered from local syncope of a finger and from chilblains. The retinal arteries were not above suspicion. In the acute stages of the disease in the left eye hæmorrhages were plentiful in the affected areas of the retina; the retina was œdematous, and I well recollect Mr. Nettleship describing its appearance as blood sodden, or boggy with blood and œdema. The conclusion which I draw on clinical grounds is to my mind sufficiently satisfying that the pathological processes which result in the appearances we know as retinitis circinata are the sequel of hæmorrhage in the outer reticular layer of the retina—whether the characteristic elliptical shape is to be explained on the limitations which Henle's layer affords I do not pretend to say. Hæmorrhages which invade the subretinal space from the outer reticular layer may organise, as Coats shows, into prominent masses of fibrous exudate; this condition may be found associated with retinitis of a circinate type.

CYST OF THE PIGMENT EPITHELIUM OF THE IRIS.

By CLAUD WORTH.

With Plate III, Fig. 1.

EVELYN F., æt. 5 years, was brought to the hospital on account of a swelling on each side of the pupil of the right eye.

History.—The condition was first noticed, in both eyes, when the child was 1 year old. The swellings have varied much in size from time to time. Last Easter those in the left eye suddenly disappeared. The child has always enjoyed good health. The eyes have never appeared red or inflamed.

The patient is the youngest of four children. The eldest squints. The other two are normal.

Present condition.—At the free edges of the pupil in the right eye are the two cysts shown in the drawing. They are faintly translucent to focal illumination. The pupil is quite free and mobile. There is no pigment in the lens capsule.

In the left eye, on each side of the pupil, is a little mass of pigment, evidently marking the sites of similar cysts which have collapsed.

Both fundi are normal. Neither eye shows any sign of past inflammation.

The point of interest in this case is that there is no reason to suppose that there has ever been any iritis.

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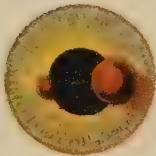


Fig. 1.



Fig. 2.



Leonardson & Co.

RUPTURE OF THE LAMINA CRIBROSA.

By MALCOLM L. HEPBURN.

With Plate III, Fig. 2, and Plate IV.

ERIC P., aged 14, came to the Royal London Ophthalmic Hospital under the care of Mr. Parsons, to whom I am much indebted for leave to publish the case.

One week previously he was walking backwards when he stumbled against the root of a tree lying on the ground, and, in turning round to recover himself, he fell, striking his left eye against a branch stump projecting from the trunk. He lost consciousness for a short time, and, on recovery, found he was unable to see with the eye, and that there was a wound about an inch long situated horizontally at the inner side of the lower lid.

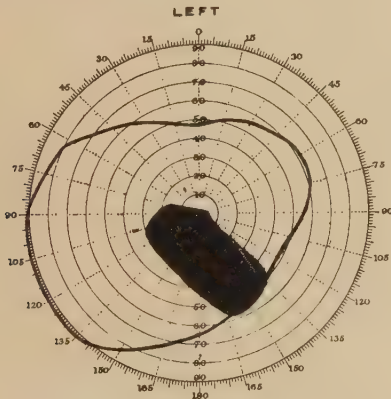


FIG. 1.

On examination, the pupils were equal and acted well to light; the vision in the right eye was 6/6, and in the left counting fingers at one metre only, on the temporal side. There was a scar on the lower lid just above the level of the lower orbital margin; but the movements of the globe were normal in all directions. The field of vision taken at this time is shown in text-Fig. 1.

By the ophthalmoscope (Plate III), hæmorrhages were visible confined to the neighbourhood of the optic disc, some close to the margin, while others were situated a little distance away. The former were flame-shaped extending up and in, while the latter were oval-shaped with well-defined borders; one such hæmorrhage was seen down and out from the disc, just beneath the macula, and another directly below, about $1\frac{1}{2}$ P.D. distant. Other small hæmorrhages were present in the same region; and the retina down and out from the disc was disturbed as in cases of commotio retinæ. In the upper temporal quadrant of the disc was a deep hole extending some distance into the optic nerve.

The whole appearance suggested the diagnosis of rupture of the lamina cribrosa, being very similar to the case described by Mr. Lang, and pictured by him in the 'Trans. Ophth. Soc.,' vol. xxi, 1901, which appears to be the only one of the kind on record.

Liebrecht, in "Klin. Monats. für Augenheilk.," September, 1909, p. 273, describes a case of evulsion of the optic nerve, and alludes to nine others already reported, but injuries of this nature are usually the result of extreme violence, and associated with severe laceration of the orbital contents:

It is an interesting speculation to consider how such an injury as is seen in the present case can be brought about.

In order to produce rupture of the lamina cribrosa, one must assume a rather violent retraction of the optic nerve from the posterior part of the globe, short of actual evulsion or injury of the nerve sheath; but it is difficult to imagine how such an injury can occur from a blow on the front of the eye which only thrusts the eyeball forcibly backwards. It might happen through a severe rotatory movement of the globe on itself sufficiently extensive to pull on the optic nerve behind. In the present case, I think it more likely that the projecting branch stump must have penetrated into the orbit beneath the globe, caught the optic nerve, and forced it backwards; thus producing the pulling action presumed to be necessary for the occurrence of such an injury.

In support of this latter contention, there is the wound in the skin at the lower margin of the orbit.

As in Mr. Lang's case, a picture taken of the fundus two months later showed considerable alterations in the appearance of the fundus (Plate IV). The hole in the disc is

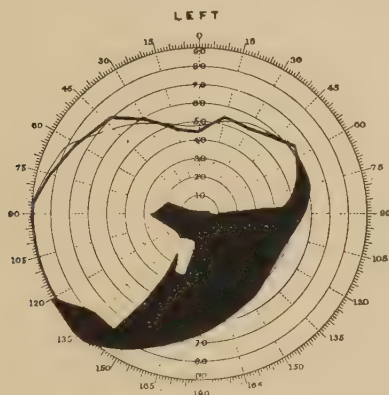


FIG. 2.

filled in by fibrous tissue; the hæmorrhages are replaced by delicate strands of fibrous tissue, some of which are also projecting from the disc itself; the retinal disturbance between the disc and the macula is much the same. There is no improvement in vision; and the field is shown in text-Fig. 2.

LITERATURE.

LANG. *Trans. Ophth. Soc.*, vol. xxi, p. 98, 1901.

LIEBRECHT. *Klin. Monats. für Augenheilk.*, vol. xlvii, September, 1909, p. 273.

THREE CASES OF POST-OPERATIVE INFECTION.

By W. ILBERT HANCOCK.

R. J., male, age 55.—Healthy man. Heart, lungs, and kidneys normal. Very septic mouth. Conjunctiva clinically normal, but no culture was taken before operation.

April 15th, 1908.—Cataract extraction under cocaine with iridectomy. Good conjunctival flap. Uncomplicated. Some soft lens remained.

A.C. re-formed within 24 hours.

April 20th.—Pillars free, pupil well dilated, capsule in wound. Eye looked extremely well, and continued to do so until April 27th (12 days after operation). The eye then became rapidly inflamed, with much ciliary and conjunctival injection. For the first few days the iris remained bright, but soon became discoloured, and much K.P. developed.

With atropine, fomentations, purging, and leeching, the inflammation quietened down, and on May 17th (a little over a month after the operation) patient was discharged, with the eye quiet, but a few spots of fine K.P.

June 10th.—He was readmitted with an acute attack of irido-cyclitis. T. full; much K.P.

With treatment as before this attack also subsided, and the eye became white; K.P. cleared up, but tension remained full, and not reduced by myotics.

June 15th.—Patient hit his eye against the locker, which started a third attack, just the same as before.

Thinking the case must be one of infection, and with a view to treating the patient with vaccine, the A.C. was tapped under the strictest precaution with a Harman's needle and a pure culture of the *Staphylococcus albus* was grown. A vaccine was prepared and the patient given 250 millions. There was no local reaction following the injection, and the eye again quietened down, but the lower part of the cornea remained partially opaque from organising deposits on the posterior surface, and the tension was +1 full.

July 8th.—As the tension remained high in spite of treatment, the capsule running up to the wound was divided with a Graefe knife. The operation induced a fourth attack of irido-cyclitis which, however, quickly subsided under treatment. Patient was given increasing doses of vaccine at intervals of 10 days, and progressed favourably until August 3rd, when he developed a fifth attack more acute than any before, with much discoloration of iris and a great deal of K.P. The eye, though improving, never quietened down completely, and on September 30th it lighted up for the sixth time with an extremely severe attack of irido-cyclitis, and large deposits on posterior surface of cornea. The iris became very vascular and somewhat drawn up, with a dense membrane in the coloboma. In spite of treatment with vaccine this attack refused to subside, and on October 16th, six months after the extraction, the eye was enucleated. Before excision the A.C. was tapped and a pure culture of *Staphylococcus albus* again grown.

Pathologist's Report (by M. S. Mayou).—"The eye was fixed in saturated Hyd. perchlor. and sections cut in paraffin and celloidin.

"Sections show the wound filled with granulation tissue (round-celled infiltration), which is necrotic in parts, and extends to the conjunctiva, which covers the surface of the wound. There is a marked fibro-vascular membrane on the surface of the iris, and a good deal of inflammatory deposit on the posterior surface of the cornea. The iris and ciliary body are much infiltrated with round-celled infiltration. Vitreous practically free from cellular exudate; choroid normal. Posterior part of globe normal. No organism stained in sections."

Bacteriologist's Report (Dr. Eyre).—"The inoculated agar tubes were covered with a profuse growth of *Staphylococcus albus* in 24 hours. Subcultures of the organism showed it to be quite typical in all its morphological and cultural characteristics. Mice inoculated subcutaneously with varying doses of the cultivation up to an agar culture emulsified in $\frac{1}{2}$ c.c. of salt solution remained unaffected. The organism may therefore be regarded as non-virulent, at any rate for the mouse."

Female, age 76.—Patient was a very robust old lady in excellent health. Heart, lungs, and kidneys normal, but very

septic mouth. She was troubled with spasmodic entropion, which had been operated upon previously; conjunctiva normal. No culture taken before operation.

In August, 1907, a preliminary iridectomy was performed, which was uncomplicated and ran a normal course.

On May 23rd, 1908, left lens was extracted; operation uncomplicated, some sticky soft lens remained.

A.C. re-formed the following day, and wound flat. Pillars of iris free; soft lens in coloboma and capsule in wound. With the exception of some conjunctival discharge, which necessitated the removal of the bandage rather earlier than usual, the eye progressed extremely well, and on the ninth day the iris and cornea were bright; slight injection around the wound, but not excessive; pupil well dilated.

On the 11th day the eye rapidly became much injected with some pain; the iris was a little discoloured, but there was no K.P. On leeching, fomenting, etc., the attack passed off; iris quite bright; no K.P.

June 22nd (a month after the extraction) an acute attack of irido-cyclitis developed; the iris was much discoloured, with vessels on the surface, and a small hypopyon. K.P. was present. Patient was given an injection of the mixed staphylococcal vaccine. This gave rise to no local reaction, but apparently upset the patient's health altogether. She began to have a temperature of 99° to 100° at night; there was a great deal of digestive disturbance, and some alarming heart failure. Although the eye improved somewhat, the hypopyon absorbing, there remained a great deal of K.P., and her general condition was far from satisfactory.

On June 30th she developed a third attack of the most acute irido-cyclitis with much K.P., the iris became very vascular, and the eye looked as bad as it possibly could be, while her general condition was still more unsatisfactory.

After a consultation with my colleague it was decided to enucleate, as it was thought the eye could never be much good as a seeing organ, and the absorption was rapidly reducing patient's strength.

July 1st.—Eye excised (six weeks after extraction). Patient made a rapid recovery.

Pathologist's Report (M. S. Mayou).—"Hardened in formol, and cut in celloidin. Sections show that the wound is not healed; the lens capsule is incarcerated within the lips. The edges of the wound show much round-celled infiltration extending beneath conjunctiva on either side. Iris and ciliary body are intensely infiltrated with polynuclear and round cells, with considerable cellular exudate in A.C. and on posterior surface of cornea. The vitreous is free from infiltration; choroid and posterior part of globe normal."

It is very striking that in each of these eyes the inflammatory changes are entirely confined to the anterior part of the eye, the vitreous and posterior part of the globe being normal.

Bacteriologist's Report (Dr. Eyre).—"From the A.C. a pure culture of the *Staphylococcus albus* was grown, and when subcultivated proved to be typical in all its various characters.

"Emulsion of an agar cultivation in sterile saline solution when inoculated subcutaneously into the mouse proved fatal for the animal in doses of 1/10th of a cultivation, the organism being recovered in pure cultivation from the spleen and heart blood; the virulence, therefore, is much in excess of that usually noticed in *Staphylococcus albus*. The vitreous was sterile."

S., male, age 70. Rather a frail old man, with normal heart, lungs, and kidney. Very septic mouth.

October 24th, 1908.—Simple extraction in left under cocaine; uncomplicated; good deal of soft lens remaining.

A.C. re-formed on the following day with circular pupil. Excellent progress until the seventh day, when the eye became very injected. This injection increased, and on November 7th (14 days after extraction) the iris was muddy, small hypopyon, pupil dilated; Tn. no K.P. With the usual treatment the attack subsided, and on November 17th the eye was practically white.

November 21st.—Developed a second attack of acute iritis, with steamy cornea; T. + 1; hypopyon and much K.P. This attack quietened down with treatment, including staphylococcal vaccines, and on December 3rd the eye was practically white. No K.P.; pupil well dilated. Tn. full.

On December 10th there was a slight flush, suggesting the commencement of another attack, but patient refused to stay in the hospital and went home to Devonshire.

On communicating with his doctor it was found that the patient, directly he got home, developed his third attack of the most acute irido-cyclitis, the chamber being $\frac{1}{3}$ full of hypopyon. With treatment the eye quietened down, but the iris remained very vascular, and the hypopyon did not completely absorb. T. + 1; no pain.

January 20th, 1909 (three months after extraction).—I saw him at Tiverton, in Devonshire, and found the eye white; no pain. Pupil small. Tn. full; hypopyon filling quite a third of the A.C.

Suspecting a *Staphylococcus albus* infection, he was given $\frac{1}{2}$ c.c. of staphylococcus vaccine. The following day he had a most violent reaction in the affected eye, and was sent at once up to town.

On re-admission the eye was intensely red; very large hypopyon filling almost half the A.C.; iris vascular and discoloured. T. + 1. Projection good; no K.P.

January 25th.—The A.C. was tapped and agar tubes inoculated with the aqueous. The A.C. was then opened below with a keratome, and washed out first with normal saline and afterwards with hydrogen peroxide, 10 vol.

The reaction after the operation was slight, and the inflammation rapidly subsided.

February 17th (third week after the last operation).—The eye was white. Tn. Cornea clear, iris of a much better colour, upper half of pupil dilated. Patient can tell the time by the watch in spite of a fairly dense capsule.

Bacteriologist's Report of the Aqueous.—A weak growth of xerosis bacillus in pure culture grew on one of the two agar tubes inoculated from the aqueous.

Clinical Type.—It is very striking that the three cases above described presented a clinical type almost exactly similar and characterised by—

THREE CASES OF POST-OPERATIVE INFECTION.

- (a) A well-marked latent period (12, 11, and 7 days respectively).
- (b) Recurrent attacks of irido-cyclitis, with intervals of almost complete absence of inflammatory reaction.
- (c) The presence of a hypopyon and extensive keratitis punctata.

Bacteriology.—Cases I and II were proved beyond doubt to be due to the *Staphylococcus albus* in pure culture. In Case III a few weak colonies of the xerosis bacillus were grown from the aqueous, but from the clinical course, and the definite reaction following the *Staphylococcus albus* vaccine, it is highly probable that the irido-cyclitis was due to the same organism as in Cases I and II. The difficulty in finding the organism was probably due to two factors: firstly, the A.C. was tapped during the intense reaction following the vaccine; and, secondly, in tapping the needle was blocked by the lymph in the A.C. and only a very small quantity withdrawn.

By most authorities the *Staphylococcus albus* is looked upon as being merely a saprophyte of the normal conjunctival sac, being found in from 79 to 96 per cent. of clinically normal conjunctivæ. Axenfeld, in his admirable treatise, asks the question: "How far should we fear an infection from the ordinary saprophytes of the conjunctiva, and to what extent has such been observed?"

He further remarks: "If we consider the common white staphylococcus of the normal conjunctiva as belonging to the class of saprophytes, we note that Gifford obtained only a slight reaction when he injected small amounts into the vitreous, often only a transient cloudiness occurred. Nevertheless I have shown that subacute cataract infection occurs in the human eye from infection by the white staphylococcus, which causes no reaction in the rabbit's cornea."

With regard to the mode of infection in these cases, one would require very strong evidence before accepting them as

being due to endogenous infection. Microscopic examination of the wound in Cases I and II is more than suggestive that the source of infection was ectogenous, although clinically in each instance the wound appeared sound, being perfectly flat, with no undue inflammatory reaction and no suspicion of pus formation.

It is true that all three cases had extremely septic mouths, and the latent period was long (12, 11, 7 days respectively), but this is of course insufficient evidence of endogenous infection.

It will be noticed that in all three cases a good deal of soft lens remained after the extraction. Now it has been shown by experimental investigation that injection of *Staphylococcus aureus* into the A.C., unless in large quantities, simply gives rise to an iritis which heals spontaneously. If, however, the lens has been freshly needled or extracted so that there is a mixture of soft lens and aqueous, it is found that panophthalmitis follows the merest trace of the culture.

There can be no doubt that eyes with a great deal of soft lens after extraction often show an alarming amount of reaction, which is generally attributed to the irritation of the soft lens matter *per se*. Personally, I believe that in most of these cases the inflammatory reaction is the result of a mild infection, and for that reason have for some time always irrigated the A.C. if the pupil after extraction is not black, and with the most encouraging results.

The advantages of irrigation are—

- (1) The removal of soft lens and therefore of a good medium for bacterial growth.
- (2) It is invaluable in simple extraction, when it is often impossible to express the soft lens matter pent up behind the iris.
- (3) It is often possible to leave the A.C. full and to wash the tags of capsule from the wound.

The objections are—

- (1) The possibility of infection, which with care should never take place.
- (2) The possibility of detaching a piece of epithelium which might be implanted in the A.C., a complication of the greatest rarity.

Vaccines were used in all three cases.

In Case I it was prepared from the aqueous, but its effect was most disappointing.

On two or three occasions there was a slight reaction and a temporary improvement, but as these intermittent attacks were characteristic of the course of the disease, it is doubtful if the vaccine did the least good.

In Case II the local effect of the vaccine was *nil*, but the injection was followed by alarming general symptoms causing no little anxiety.

In Case III the first few injections apparently had no effect at all, but after the last injection the local reaction was excessive and the hypopyon much increased.

With regard to local treatment, the brilliantly successful result of washing out the A.C. in Case III would encourage me to do it again in similar cases and not to delay it too long. Had the A.C. been washed out with hydrogen peroxide in Cases I and II it is more than likely they would have recovered with useful vision.

TERATOMA OF THE ORBIT.

By R. J. COULTER and G. COATS.

With Plates V to X.

THE rarity of the following case, and its interest as well from the ophthalmological as from the pathological standpoint, constitute our excuse for recording it.

On March 2nd, 1909, I (R. J. C.) was asked by Drs. Haslett and Elliot of Pontypool to see an infant suffering from exophthalmos. They stated that the child was born on February 27th after a normal labour, no instruments being used, and it was at once noticed that the left eye was pushed forwards out of the orbit. On making a closer examination it was found that the cornea was clear but looked flat and the lens was opaque and yellow. When seen by me on March 2nd, the eye was pushed straight forwards so that it lay outside the orbit with the eyelids stretched around its equator; the cornea was opaque and a tense soft tumour could be felt in the orbit. The child was admitted to the Pontypool and District Hospital, and on March 7th I saw it again, when the proptosis was more marked: the cul de sac was obliterated and the conjunctiva was stretched straight from the edge of the lids over the eyeball. The conjunctiva and cornea were ulcerated from exposure, and the skin was stretched so that the external canthus was half an inch in front of the external orbital margin.

The child was failing owing to the irritation from the eye, and it was felt that something ought to be done to relieve the condition, but a radical operation seemed almost certain to be fatal; I therefore tapped the most prominent part of the orbital cyst with a hypodermic needle, and removed about 3 drachms of blood-stained fluid.

This relieved the tension sufficiently to allow of the eyelids being brought together over the eye; their edges were made raw and sutured, in the hope that they might become united, and exert enough pressure to prevent the cyst from refilling. As was to be expected, however, the sutures cut out, and the

cornea sloughed. The child remained in the hospital until March 17th when it died of exhaustion.

Through the kindness of Dr. Haslett I was enabled to make a partial *post-mortem* examination on March 18th, when a tumour was found in the left orbit situated inside the cone of muscles. This was removed and sent to Mr. Coats for examination. The walls of the orbit and the contents of the cranial cavity were normal.

Pathological Examination. (Plates V to X.)

The specimen was fixed in formalin; it was divided through the middle by a vertical antero-posterior section. It consists of an egg-shaped tumour with the remains of the eye at one end (Plate V, Fig. 1, A), and measures, exclusive of the eye, 2.5 cm. antero-posteriorly, 2.2 cm. vertically, and about the same transversely. The cornea has sloughed, and the iris, lens, and vitreous have disappeared; some remains of the ciliary body are present; the choroid is thickened and the retina is indistinguishable. The tumour is lobulated, but has a smooth surface on which the external muscles are recognisable; its connection with the walls of the orbit must have been loose. It is firmly adherent to the posterior surface of the globe, but is sharply marked off from the sclera, which is quite intact. The optic nerve (Fig. 1, B) runs through the midst of the tumour: it is not invaded and is only slightly displaced, being a little bowed downwards in its posterior part. On the cut surface the most noteworthy features are a number of plaques of hyalin cartilage (Fig. 1, D) and numerous cysts (Fig. 1, C) of variable size; they are often lobulated, or multilocular with free communication between the loculi; an especially large one occurs above; the lining membrane is smooth and sometimes blood-stained. These cysts are empty, but there are others of small size in the anterior part of the tumour which are filled with a stiff greenish yellow substance (Fig. 1, E). The tissue around these cysts is condensed, and shows yellow streaks of adipose tissue.

Microscopical Examination.

In order to make the sections of convenient size, one half of the tumour was divided transversely into an anterior portion

PLATE V.

FIG. 1.— $\times 1 \cdot 25$. Macroscopic view of the divided tumour. A, eyeball; B, optic nerve; C, cyst; D, hyalin cartilage; E, a cyst with greenish semi-solid contents.

FIG. 2.— $\times$ about 5·5. Anterior half of the tumour. A, the eye; D, hyalin cartilage; F, ossification in cartilage (see Fig. 4); E, cyst lined with intestinal mucous membrane (see Fig. 7); G, cyst lined with brain substance (see Fig. 6); H, lymphoid tissue.

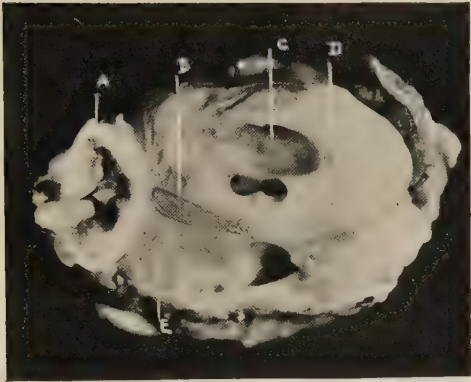


FIG. 1.

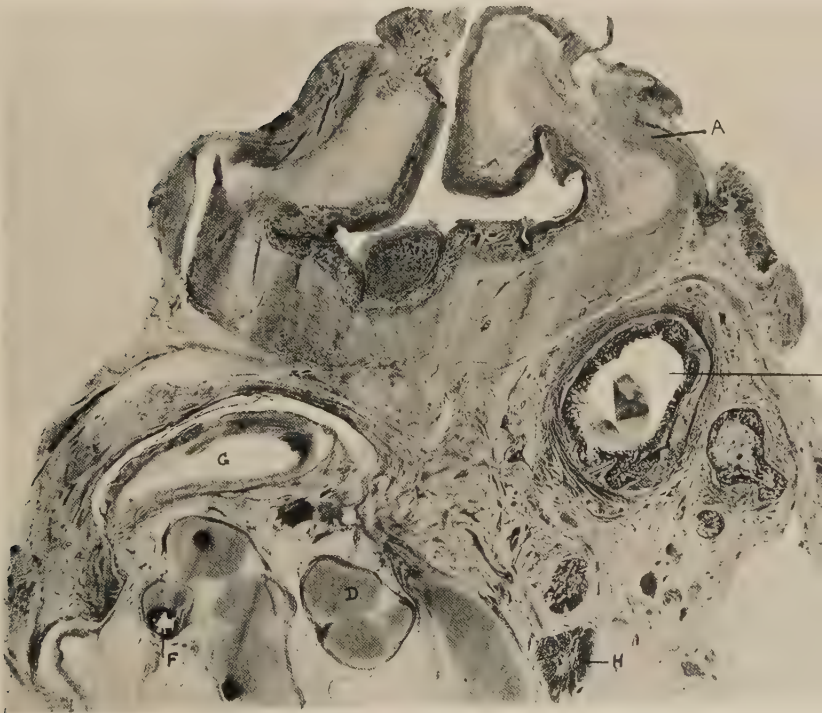


FIG. 2.

(including the globe (Fig. 2)) and a posterior (Fig. 3). An antero-posterior slice about 0·5 cm. thick was taken and cut in serial section, every fifth being mounted. In addition, pieces from various other parts of the tumour were cut. It is believed that no essential tissues could have been missed in the histological examination.

The anterior parts of the globe (Fig. 2, A) have sloughed away except for a small piece of conjunctival epithelium and of ciliary muscle. The lens is absent. The retina is indistinguishable among round cell infiltration. The choroid is much thickened and congested, and is densely infiltrated with lymphocytes, plasma cells, and polymorphonuclear leucocytes. The sclera is thickened and folded. The nerve is possibly slightly atrophied, but is normal in its general architecture; the central artery and vein are present, and there are many vessels in the trabeculæ.

The tumour consists of a jumble of tissues derived from all three embryonic layers. The following are represented:—

- (1) Connective tissues of various types.
- (2) Skin (Plate VI, Fig. 3, K; Plate VII, Fig. 5).
- (3) Brain substance (Plate V, Fig. 2, G; Plate VIII, Fig. 6).
- (4) Intestine (Plate V, Fig. 2; Plate VIII, Fig. 7).
- (5) An internal gland, (?) Liver (Plate IX, Fig. 8).
- (6) Respiratory mucous membrane (Plate IX, Fig. 9; Plate X, Fig. 10).
- (7) Cysts with linings other than intestinal or respiratory mucous membrane (Plate X, Figs. 11 and 12).

(1) *Connective tissues*.—These are not embryonal in type, but have attained a degree of development corresponding on the whole with the age of the child. The following are represented: (a) A somewhat cellular fibrous tissue which forms the groundwork of the whole tumour. Round most of the cysts, and in places elsewhere, it undergoes condensation. It contains well-formed vessels and nerves. (b) Adipose tissue (Fig. 3, L). This occurs in island-like areas among the fibrous tissue. In places it is fully formed; elsewhere more cellular than adult adipose tissue, and with cells incompletely distended with fat. It is probable that these two are not tissues strictly proper to the

PLATE VI.

FIG. 3.— $\times$ about 5.5. Posterior half of the tumour. C, cyst lined with a single layer of epithelium. The wall is papillated in places (see Fig. 12) ; D, hyalin cartilage traversed by tracks of loose fibrous tissue, containing vessels. The deeper staining of the cartilage in this section as compared with Fig. 2 is merely due to a difference in microscopical technique ; J, a cyst lined with stratified ciliated epithelium (see Fig. 10) ; the wall is papillated on the right side ; K, a cyst lined with skin (see Fig. 5) ; L, orbital adipose tissue ; M, rectus muscle.

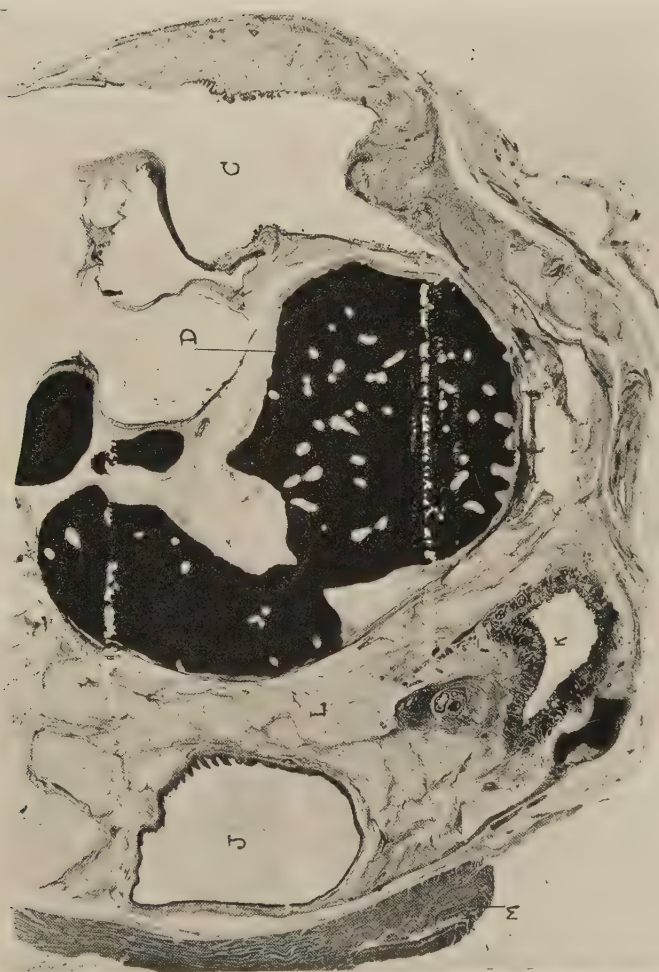


FIG. 3

tumour, but in part at least represent the original tissues of the orbit. (c) Lymphoid tissue (Fig. 2, H). This occurs in considerable isolated nodules in various parts of the tumour. It shows no features worthy of note. (d) Hyalin cartilage (Figs. 2 and 3, D, Fig. 4). Large areas of this tissue were already visible with the naked eye. In the anterior part of the tumour the cartilage is mostly avascular; posteriorly, where larger plaques occur, it is traversed by channels containing vessels embedded in a loose connective tissue. The cartilage is rather richly cellular in type, and on its surface the connective tissues are condensed to a thick perichondrium. In places the matrix is calcified, and there are several patches of true ossification in cartilage (Fig. 4) which show all the usual phenomena associated with that change—enlargement and vertical grouping of the cartilage cells, irruption of osseous material and blood-vessels from the periosteum, etc. (e) Bone. In addition to the areas of ossification just mentioned an isolated bony tube occurs. It is surrounded by thick periosteum, and has a well developed vascular marrow, containing, *inter alia*, many eosinophile cells. Although its cylindrical form suggests an attempt to form a long bone, it is very much smaller than even the phalanges of a newly born child.

(2) *Skin* (Fig. 3, K, Fig. 5).—This is found in at least two distinct and isolated patches. In both instances it completely lines a small cavity, containing some epithelial *débris*. In the section shown in Fig. 3, the cavity is simple; elsewhere it has a more complicated form, and the surface of the lining skin is consequently very extensive. The epithelium shows a fairly well defined stratum Malpighi, but a stratum granulosum and stratum lucidum are absent; cornification is present on the surface. The corium is papillated, and contains very numerous hair follicles—much more numerous than would be found in any but the most hairy parts of the body. The follicles are usually small and comparatively superficial; some, however, are larger, and are deeply implanted in the subcutaneous adipose tissue; many are dilated, the hair lying loosely in the middle of the cavity. Sebaceous glands are present but are not very large. A very few ill-developed sweat glands are found in the deeper layers of the corium.

PLATE VII.

FIG. 4.— $\times 90$. Ossification in cartilage.

FIG. 5.— $\times 75$. Skin. The hair follicles are very numerous and very irregularly disposed. Some of them are dilated, the hair being seen loose in the cavity. Sebaceous glands are small at this place, and sweat glands are not shown. The epithelium shows no proper stratum granulosum, etc.

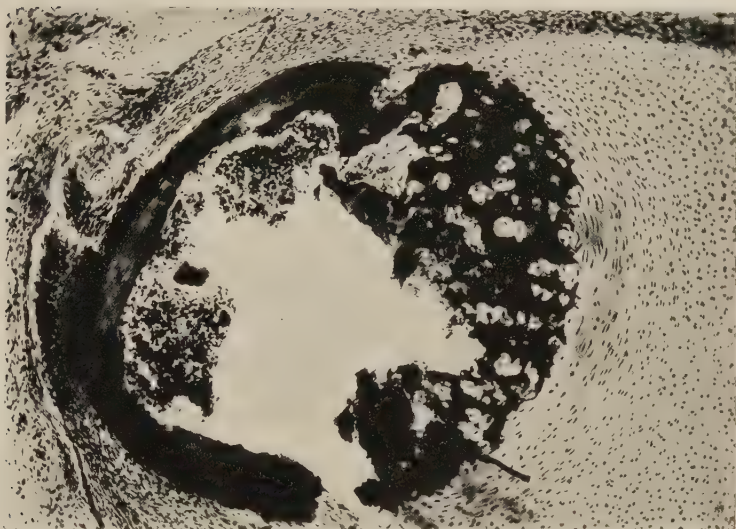


FIG. 4.

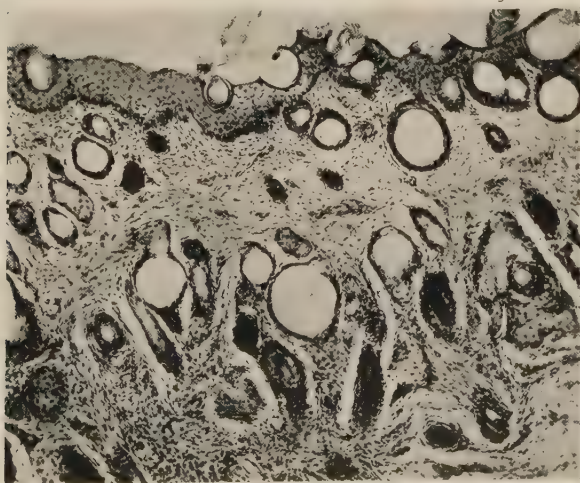


FIG. 5.

(3) *Brain Substance* (Fig. 6).—Comparatively large tracts of this tissue occur in the anterior half of the tumour, where it lines a number of narrow spaces. It consists of a delicate reticular ground-work, containing nuclei of various shapes and sizes. On staining with v. Lenhossék's thionin blue method some of these cells have the characteristic shape of ganglion cells, but only a comparatively few ; for the most part the nuclei probably belong to the neuroglial system. Part of the surface is lined by a single layer of vertical cells, which in places resemble an epithelium ; elsewhere, however, they send fine threads off into the reticular tissue beneath, and they seem to be merely a specially arranged and more closely packed layer of that tissue. It is probable, therefore, that they represent the condensed neuroglia (ependyma) which normally lines the ventricles, rather than the ventricular epithelial lining. This is further confirmed by the fact that the cells are not ciliated. A few delicate blood-vessels traverse the tissue.

(4) *Intestine* (Fig. 2, E, Fig. 7).—There are several isolated patches of this tissue, corresponding to the cysts filled with a "stiff greenish substance" mentioned in the macroscopic examination. Not merely the mucous membrane, but the whole thickness of the intestinal wall is represented. The mucous membrane consists of a number of tubular glands, surrounded by a richly cellular vascular tissue. Their arrangement is perhaps rather less regular than in the normal intestine ; otherwise they imitate it perfectly. The lining cells are almost entirely goblet cells ; the glands therefore correspond to those of the large intestine. Towards the surface the cells are not infrequently broken down, and the interior of the cysts is filled with mucous contents, with here and there a few groups of nuclei. On the external aspect of the glands there is, in places, a well-defined muscularis mucosæ ; more commonly they rest directly on a rather cellular fibrous tissue, without the intervention of a muscular layer. Outside this fibrous tissue comes a thick layer of involuntary muscle, which is not always separable into distinct strata, but in typical situations can be divided into an inner layer of longitudinal nuclei, and an outer of transverse ; this arrangement would correspond with a transverse section of the intestine. In some situations there is

PLATE VIII.

FIG. 6.— $\times 120$. Brain tissue. It consists of a delicate reticular ground-work with nuclei of various shapes and sizes. Ganglion cells are not distinctly recognisable here. The vertical cells lining the cyst cavity (? ependyma) are seen.

FIG. 7.— $\times 90$. Intestinal mucous membrane. Tubular glands lined with mucous cells are seen above. They are surrounded by a cellular connective tissue. Two layers of involuntary muscle are seen below the inner being cut longitudinally, the outer transversely; in places there was another layer cut longitudinally outside this again.



FIG. 6.

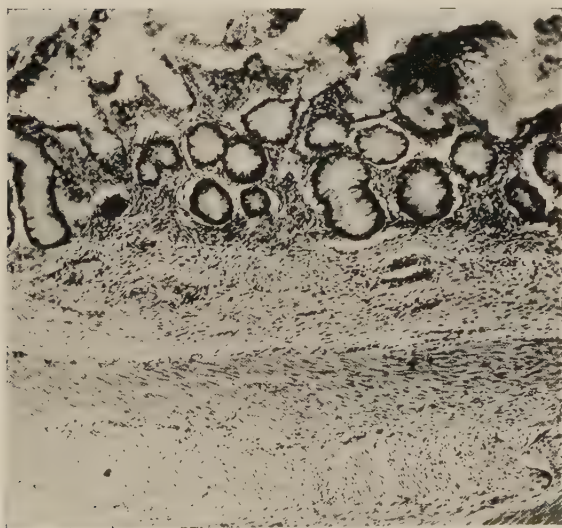


FIG. 7.

another thin longitudinally cut layer outside the transverse, while on the contrary in other cysts the muscular tissue is but poorly developed.

(5) *An Internal Gland* (Fig. 8).—There is only one small patch of this tissue. It consists of an irregular system of epithelial strands, interpenetrated by a highly cellular and fairly vascular connective tissue. For the most part the strands seem solid, but in places the cells surround a minute circular lumen (A). The individual cells are not very well demarcated from one another; they have a medium sized, darkly stained nucleus, and a large, faintly granular protoplasmic body. The whole mass is surrounded by cellular connective tissue. Although situated near some patches of intestine it seems to have no connection with them. The most probable explanation would seem to be that this is a piece of ill-developed foetal liver, or it may possibly be suprarenal. This interpretation is given with diffidence, however, as it is usually asserted that these tissues do not occur in tumours of this type. It might also possibly be thyroid, which is sometimes found in these tumours.

(6) *Respiratory Mucous Membrane* (Fig. 3, J, Figs. 9 and 10).—There are at least two cysts with a lining of this type. One of these is comparatively smooth walled. The epithelium consists of from three to four layers of rather low cells, the innermost being provided with a fringe of short cilia; in certain limited areas the cilia are absent. Externally the epithelium rests directly on a fairly dense fibrous tissue which, in its outer layers, becomes looser and contains large racemose glands. These glands are not very well formed, the loculi being comparatively few in number, and separated by a considerable quantity of cellular tissue. The gland cells are vertically elongated, and have a clear protoplasm; the nucleus is situated towards the external border. They are probably ill-developed mucous glands. No cartilage is present in the actual cyst wall, but in its general characters the mucous membrane agrees with that of the upper air passages, and particularly with the tracheal.

The other cysts lined by respiratory mucous membrane have a thicker epithelium (five to six layers), the superficial cells are much more columnar, and the fringe of cilia is at once more continuous and broader; mucous cells occur among the ciliated.

PLATE IX.

FIG. 8. — $\times 120$. A tissue consisting of epithelial strands surrounded by a richly cellular, vascular connective tissue. At one place above, and to the left, a minute lumen is visible, surrounded by epithelial cells (A). The tissue most probably represents fœtal liver.

FIG. 9. — $\times 75$. Respiratory mucous membrane. The lining epithelium is ciliated, but the magnification of this figure is insufficient to show this. In the deeper layers of the mucous membrane a mass of rather atypical glandular substance is present. Cartilage is not present in the wall.

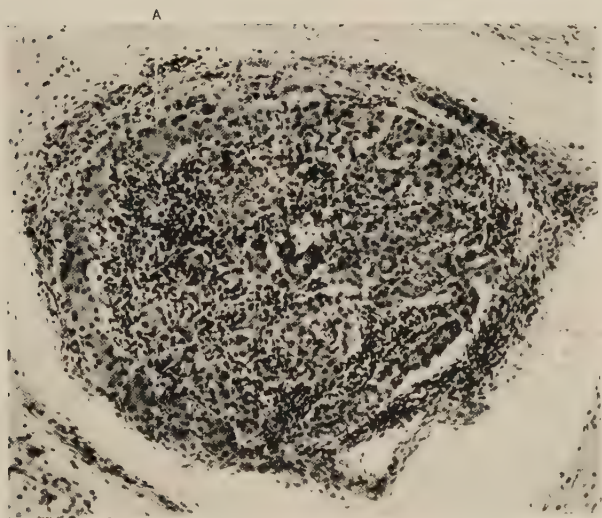


FIG. 8.

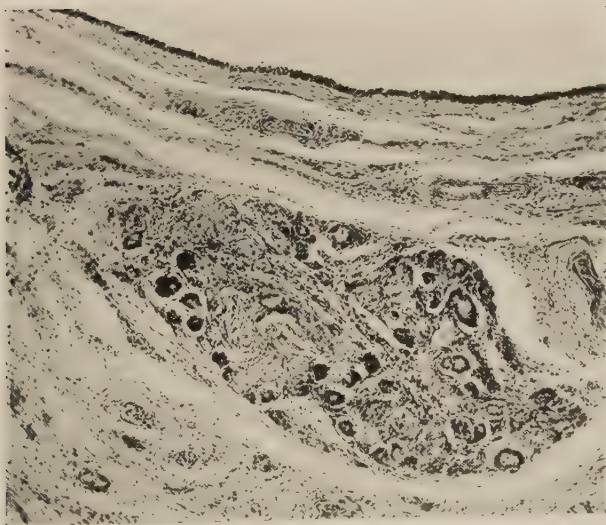


FIG. 9.

The epithelium rests directly on the fibrous wall of the cyst, which is thrown into considerable plications or papillæ in places. A few typical mucous glands are present. This tissue corresponds in all respects with the respiratory portion of the nasal mucous membrane.

(7) *Other Types of Cysts.*—Under this heading are included a number of cavities lined with epithelia not sufficiently characteristic for systemic classification. Such are the following:—(a) There is one cyst in the anterior half of the tumour lined by a stratified squamous epithelium (Fig. 11). The cells of the outermost layer are small, closely packed, and deeply stained; those of the middle layers are larger and paler, with well-defined cell limits; the innermost cells are of the same kind, but slightly flattened. The epithelium is surrounded by, and rests directly upon, a broad area of the densest, least cellular, fibrous tissue which occurs anywhere in the tumour; no glands or downgrowths are present; the epithelium varies considerably in thickness, but where cut vertically it is usually six- to eight-layered. Allowing for minor differences due to the fact that it clothes an enclosed cavity, not a free surface, one might compare this epithelium with that of the conjunctiva, the canaliculi and lacrymal sac, the mouth, pharynx and œsophagus, the vagina, etc.; it would be unsafe to attempt to classify it more particularly.

(b) There are many cysts lined with a single layer of cubical or flattened cells (Fig. 3, C, Fig. 12). The largest cysts are included in this group, and they are frequently multilocular. Usually the wall is quite smooth, but in places curious little complex papillæ occur (Fig. 12). In a few situations the epithelium shows slight pigmentation, and groups of round pigmented cells occur in the stroma. These cells have no resemblance to the hexagonal pigment, or to the chromatophores of the uveal tract; they do not represent therefore a rudimentary eye such as has been described by v. Hippel in his case of orbital teratoma (see later), and by other observers in similar tumours in the ovary and elsewhere. Having in mind the "blood-stained fluid" withdrawn from the cysts by operation (see p. 64), and the fact that small hæmorrhages occur here and there in the cyst walls, there can be little doubt that the pigment is of hæmorrhagic origin.

PLATE X.

FIG. 10.—Stratified ciliated epithelium lining the cyst marked J in Fig. 3.

FIG. 11.— $\times 120$. Stratified squamous epithelium lining a cyst. It rests directly on dense fibrous tissue, containing no glands.

FIG. 12.— $\times 120$. Papillæ in the wall of the cyst marked C in Fig. 3.
Where the epithelium is cut vertically it consists of a single layer of cubical or flattened cells.

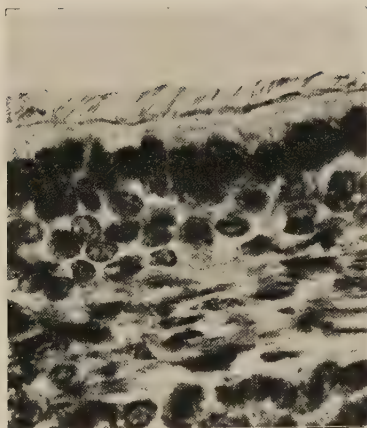


FIG. 10.



FIG. 11.

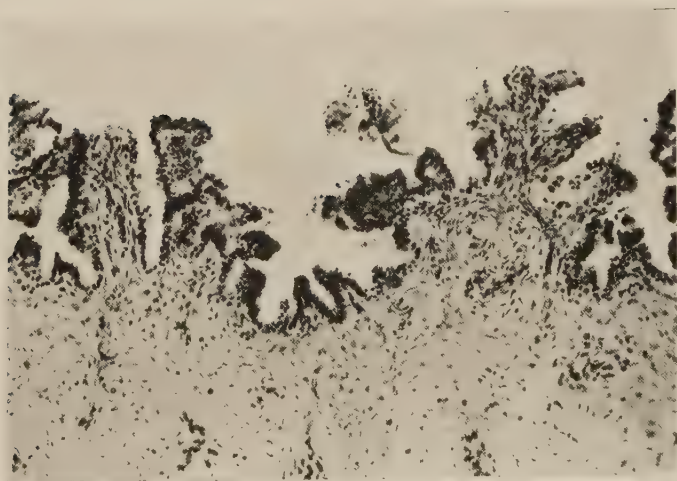


FIG. 12.

(c) In most cases only one kind of epithelium is found in any individual cyst; one or two cavities, however, are lined partly with a stratified incompletely ciliated epithelium five to six layers thick, partly with a single row of mucous cells. The transition from one to the other is usually quite abrupt, and the epithelium may alternate between the two types more than once within the high power field of the microscope.

All the tissue above described are massed together in inextricable confusion—the respiratory and intestinal mucous membranes, for instance, are in close proximity. On its outer aspect the tumour has no proper delimiting capsule; it passes off indefinitely into the surrounding orbital tissues. The superior and inferior rectus muscles pass over its external surface without being invaded; structurally they are normal.

After the above description the diagnosis of “teratoma of the orbit” requires no further justification. In view of the extreme rarity of the condition, and of the fact that no case has been reported in English literature for the past 25 years, it seems worth while to give a brief summary of all the known instances. An admirable *résumé* of the whole subject will be found in v. Hippel’s paper, to which acknowledgment is here expressed.

Case of Holmes (Trans. Path. Soc. Lond., vol. xiv, p. 248, 1863).—This is a possible, but very doubtful instance. The diagnosis rests partly on Holmes’s own description, but more on the fact that in discussing Lawson’s indisputable case, Holmes’s was referred to as being of a similar nature. The pathological examination is very incomplete.

Female, æt. 7 weeks, twin. Congenital, rapidly growing, lobulated tumour of R. orbit, extending also on to malar and upper jaw bones; cystic; hard; separable from the bones of the orbit; moveable by the ocular muscles. Eye protruded at birth; became ulcerated and perforated. Excision of tumour; recovery; no recurrence at least for some time afterwards. Pathologically the cut surface of the tumour much resembled udder; many cysts containing thin serum. Microscopically the solid portions consisted of “fibrous tissue and simple nuclei.”

Case of Bröer and Weigert (Virch. Arch., vol. lxvii, p. 518, 1876).—This is the first indubitable case.

Child, æt. 1 day. Tumour of R. orbit displacing nose, cheek, and corner of mouth; projecting through and constricted by eyelids; fluctuation present. Movements observed in tumour along with those of the other eye. Globe present on top of tumour; cornea slightly opaque. Five days later tumour had grown; surface eroded. Hypopyon in anterior chamber. The cyst was punctured, and clear yellow fluid escaped to such an amount that the tumour shrank to one-half. Excision of tumour; death two days later from pericarditis.

Pathological Examination.—Tumour is the size of a small apple; pyramidal in shape, with base towards the globe. Contains many cysts with firm fibrous walls; one of these cysts has the appearance of a loop of intestine. Islands of dense fibrous tissue, cartilage and bone are present. Microscopically: adipose and fibrous tissue; collections of round cells; cartilage; ossification in cartilage; bone with marrow; involuntary muscle; vessels; elongated tubes of epithelium. Cysts lined with three types of epithelium:—(1) Small cysts with stratified squamous epithelium, deepest layers round or cylindrical, most superficial cornified; pearly cornified epithelium in cavity; solid epithelial downgrowths (rudimentary sebaceous glands) into subjacent tissues. These cysts are evidently epidermal. (2) Cysts lined with single layer of cylindrical epithelium; in long tubes or round cavities. In the intestine-like structure there are regular tubular glands in the form of Lieberkühn's crypts; they rest upon tough connective tissue which is followed first by looser tissue and then by smooth muscle in an inner circular and an outer incomplete longitudinal layer. (3) Cysts lined with stratified ciliated epithelium. Cartilage in underlying fibrous tissue which also contains glands. The larger cysts have a smooth fibrous wall lined by stratified ciliated epithelium. These cysts represent bronchial mucous membrane.

Case of Ahlfeld (Die Missbildungen d. Menschen, I. Abschnitt, Taf. VI, Fig. 11, p. 52. Leipzig, 1880).—This case differs from the others in being externally recognisable as a separate individual.

From the left orbit of a large, well-developed child there projected the buttock and left leg of a second individual. Near the buttock there was a little proboscis like the proboscis of a cyclops. In the line of the maxillary cleft there was a tumour of liver-like consistence, and there was a large frontal encephalocele.

Case of Lawson (Trans. Path. Soc. Lond., vol. xxxv, p. 379, 1884).—Male, æt. 2 days, born at full term and well grown. Parents healthy. Complete exophthalmos R. Cornea dull and covered with mucus. Eye excised, but not the tumour. Died three months later with convulsions and coma. P.M., basilar meningitis. Tumour seemed to have originated in body of sphenoid and grown into orbit; four to five cysts projected into cranial cavity. Tumour contains many multilocular cysts. Microscopically: hyalin cartilage; many cysts lined with pavement epithelium; some of the smaller filled with cells undergoing colloid degeneration. More solid parts consisted of masses of embryonic round and spindle cells, and more fully developed fibrous tissue. Evidence of formation of glandular tissue in several situations. Lawson believed that the tumour was of embryonic origin, and that an attempt was being made to produce glands and the lower forms of connective tissue. He pointed out that it contained elements formed from more than one blastodermic layer, and therefore sought to derive it from the pituitary gland, in which also more than one embryonal layer plays a part. In the discussion, Marcus Beck and Rickman Godlee named it a teratoma, and drew attention to its resemblance to Holmes's case.

Case of Courant (Centralbl. f. Gynäkologie, vol. xvii, p. 740, 1893).—New-born child, normal except for the orbit. Left eye protruded 2 cm. by a tumour which fills the whole orbit and is separated from the globe by a shallow furrow. Cornea opaque; hypopyon. Tumour of hard, elastic consistence; slight movements along with these of the other eye. Exenteration second day; recovery. Tumour is the size of a small apple; attached to globe and surrounds nerve; lobulated surface; in places contains cysts with serous, blood-stained, or mucous contents;

plaques of bone, especially in the centre. Microscopically: fibrous and adipose tissue; hyalin cartilage; ossification in cartilage; large cysts lined by cylindrical epithelium; mucous cysts with stratified cylindrical epithelium sending down processes into subjacent tissues; in deeper layers probably involuntary muscle. Another cyst has a low cylindrical epithelium and glandular acini, with long ducts, in the underlying tissues. Hair follicles are found.

Case of Ewetzky (Moscow ophth. Soc., 1904. Wratsch, vol. xxi, p. 690. Nagel's Jahresbericht, 1904, p. 303).—This case is somewhat doubtful. Child, æt. $4\frac{1}{2}$ months. Smooth, elastic, immovable tumour of R. orbit. Rudimentary eye at the apex. After exenteration the remains of the tumour began to grow again. Microscopically: connective tissue; adipose tissue; bone; cartilage; nervous substance; pigmented tissue; cysts, some lined with ciliated epithelium. Ewetzky calls the tumour a "cephaloma orbitæ," and believes it to be a hernia cerebri in which the communication with the brain had become cut off.

Case of v. Hippel (Arch. f. Ophth., vol. lxiii, p. 1, 1906).—Child, æt. 5 days, otherwise normal. Great exophthalmos. Cornea infiltrated; iritis. Tumour the size of a medium apple; optic nerve runs through the middle of it. Microscopically, tumour contains:—(1) Central nervous tissue in the form of cysts lined with a tissue composed of nerve fibres and neuroglia. In places papillated structures like the choroidal plexuses. (2) A rudimentary optic vesicle, turned outside in, so that the retina is outside and is inverted; the pigment epithelium comes next, and the choroid is innermost. No lens vesicle. v. Hippel supposes this peculiar arrangement to arise as follows: a primary optic vesicle was formed, but, meeting with no lens vesicle, it was not invaginated from in front; subsequently, however, it was invaginated from behind by the growth of the tissues of the tumour. (3) Cysts clothed with epidermis and containing hairs and sebaceous glands in their walls. (4) Hyalin cartilage. (5) Bone with marrow and osteoblasts. (6) Involuntary muscle. (7) Necrotic areas with chalky deposits. (8) Large hæmorrhages, especially posteriorly. (9) Distended vessels, giving in places

the appearance of a cavernous angioma. (10) Cysts lined with high cylindrical epithelium containing mucous cells in places. They probably represent part of the digestive or respiratory tracts. In certain areas there is a resemblance to intestinal villi. (11) Glandular tissue of an atypical kind, not unlike foetal thyroid. (12) Lymph follicles. (13) Atypical epithelial structures, with a resemblance to carcinomatous tissue. No definite organs were present.

Case of Mizuo (Ber. ü. d. 35ste Versamml. d. ophth. Gesellsch. z. Heidelberg, 1908, p. 347).—This is a very curious case. In a Japanese child at birth there was a tumour of the left orbit, which rapidly increased in size. Punctured 2nd day—opaque, watery fluid escaped. On 40th day again punctured, when a brown purulent fluid escaped. This opening did not close, and on the 50th day a left foot became protruded from it, and presently a whole foetus was expelled, the process being “very like a real delivery.” The foetus remained connected with the deeper parts of the orbit by the umbilical cord, which still passed through the opening in the skin; “it appeared as if a little monkey were creeping on the left side of the face.” Foetus well formed; covered with fine hairs; long hairs near the head; but the head itself denuded of skin, its surface consisting of a spongy, yellow-grey, brain-like substance. Rudimentary arms; well-formed nates and legs, the toes and nails being well developed. Connected with the thigh and lower part of the belly, a penis-like structure, with a Y-shaped slit at its apex.

The child (autosite) showed no other abnormalities. Globe normally formed, displaced forwards and upwards, defective movement downwards; disc somewhat atrophic. The umbilical cord was divided between ligatures and the remains of the cord subsequently excised; the opening in the skin gradually closed, and the child made a good recovery, and seemed to have some vision in the left eye.

Pathological Examination.—Foetus weighed 68·3 grams. Brain substance, with ganglion cells, present in head; cerebellum distinguishable. Ill-formed vertebræ, spinal ganglia, voluntary muscle and lymph glands present. Anteriorly at base of skull there was a cavity representing mouth and pharynx, lined with

ciliated epithelium, and with glands like salivary glands opening into it; continuous with this cavity, spaces lined with gastric, duodenal, and intestinal mucous membrane, and clothed with well-formed layer of muscle. Large intestine also represented. Spleen present. Representatives of primitive kidney, prostate, urethra, etc. Skin normal.

Mention must also be made here of certain tumours of the orbit which contain brain substance. Some of these are ordinary encephaloceles, and have no connection with the cases now under discussion; they are presumably caused by protrusions from the cerebral vesicle in early foetal life. There are others, however, which are either confined to the orbit, or, if they pass within the skull, have no connection with the brain proper. Some of these also are no doubt encephaloceles in which the connection with the brain has become nipped off; but when the tumour itself is large, and the brain is free from abnormalities, this origin is impossible. A remarkable case of the kind has been reported by Parsons and Coats: a large mass of highly developed brain substance, containing abundant ganglion cells, was present partly in the orbit and partly within the skull; although it pressed directly upon, and displaced, the base of the brain, there was no connection between the two, and the brain was quite normally formed. Since there was no defect of the brain, it is evident that the tumour was not an encephalocele in the ordinary sense: it must have arisen in early embryonic life, presumably by the segregation of certain cells of the developing brain, their replacement by other brain cells, and their subsequent independent growth (see p. 88). In this sense the tumour was a teratoma, but it lacked an element of the teratomata in the strictest sense—namely, derivatives of hypoblast (see p. 84).

A somewhat similar observation is that of Lagrange (*Arch. d'Opht.*, vol. xv, p. 536, 1895). A congenital, rapidly growing tumour of the right orbit was found to contain connective tissue and embryonal brain substance; the tumour contained some degenerative cysts, and passed through the sphenoidal fissure and into the maxillary sinus. Allied cases are recorded by v. Duyse and Talko (*Encyclop. Franc. d'Opht.*, vol. ii, pp. 558 and 560, 1905), and by Vanzetti (*Annali di Ottalm.*,

vol. xxx, p. 33, 1901), and there are no doubt other cases in literature. Instances in which cerebroid tissue occurred within the globe itself are reported by Krückmann (*Arch. f. Ophth.*, vol. xlvii, part i, p. 50, 1898), and by de Waele and Lewuillon (*Encyclop. Franc. d'Ophth.*, vol. ii, p. 561, 1905); the best explanation is probably that of the last two authors, who suppose that the retina has erred in development and gone back to the type of its parent tissue—the cerebral vesicle; indeed, something of the kind occurs in most cases of microphthalmos with cyst. Sacral tumours consisting largely of brain substance have been reported by Borst (*Ziegler's Beiträge*, vol. xxxi, p. 419, 1902) and by Hennig (*Ziegler's Beiträge*, vol. xxviii, p. 593, 1900).

In a tumour which had otherwise the character of a dermoid, and which was attached to the anterior part of the globe, Wagenmann found a cyst lined by ciliated epithelium, and with cartilage in its neighbourhood (*Arch. f. Ophth.* xxxv, 3, p. 111, 1889). It is doubtful if these characters are sufficient to earn his case a place among the true teratomata. Cartilage is not very infrequent in ordinary superficial dermoids of the cornea and conjunctiva, and it is possible that the acquisition of cilia is not beyond the epithelial powers of metamorphosis. On the other hand, the association of ciliated epithelium and cartilage is highly suggestive of respiratory mucous membrane, the occurrence of which, in conjunction with dermal elements, would satisfy the essential diagnostic requirements of a true teratoma—namely, the representation of all three blastodermic layers. A cyst lined by ciliated epithelium has been found in a congenital anterior staphyloma by Krükow (*Arch. f. Ophth.*, vol. xxi, 2, p. 226, 1875); he explained it as due to displacement of respiratory epithelium, and its inclusion in the corneal cicatrix, a hypothesis which might possibly be extended also to Wagenmann's case.*

* A tumour described as "teratoid" by Rothschild (*Deutsche med. Wochenschr.*, p. 2048, 1907) is certainly different from those here reported. It first appeared in an adult (man, æt. 28), was removed by a Krönlein's operation, and showed microscopically a ground-work of mucoid and fibrous tissue with cartilage in places, traversed by tubes and solid processes of cells of epithelial type; in places there were cysts with papillated walls, elsewhere islands of cornified epithelium with a laminated arrangement. This neoplasm was probably either a mixed tumour such as occurs in the parotid,

The cases of true teratoma here cited group themselves naturally into those in which the members of a second individual were externally recognisable (cases of Ahlfeld and Mizuo), and those in which there was only an amorphous tumour mass (cases of Bröer and Weigert, Lawson, Courant, Ewetzky, v. Hippel and the present case). It is common to both groups, however, that structures derived from the epi-, meso-, and hypo-blast are present. This is the distinguishing mark of the true teratomata. The name has also been applied more loosely and broadly to any tumour which consists of tissues foreign to the part in which it is situated, a definition which would include the dermoids, as well as such tumours as chondromata of the testicle, etc. A tumour of this type, if it contained elements derived from only one embryonic layer, might have arisen in later foetal life by the dislodgement of already differentiated cells from their own layer, and their implantation in a foreign tissue. The same explanation might hold for tumours containing either epiblast and mesoblast, or hypoblast and mesoblast, since the mesoblast is in the closest relationship with both, and would very probably share in their dislodgement; but it is scarcely tenable for tumours which contain epiblast and hypoblast, since these two layers are scarcely anywhere in contact, and still less is it tenable for tumours containing derivatives of all three layers. The latter type of tumour must have originated in the faulty development of an ovum, or of its daughter cells before differentiation was advanced, and it seems best to restrict the term "teratoma" to this group; its relations to the others will be discussed presently.

or one of those orbital tumours of uncertain origin, which are variously described as adeno-carcinomata of the lacrymal gland, endotheliomata, cylindromata, etc.; in these a tubular structure is not uncommon, and structures like cell nests sometimes occur. The name "Terato-neuroma" is used by Verhoeff (*Trans. Amer. Ophth. Soc.*, vol. x, pt. 2, p. 351, 1904) to describe a complex tumour arising from the pars ciliaris retinae; Alling (*Trans. Amer. Ophth. Soc.*, vol. x, pt. 2, p. 265, 1904) has used the name "teratoma" for an intraocular tumour of doubtful nature, described by him in a child aged 4 years. Neither of these observations has any bearing on the present subject.

Leaving out of account for the moment the cases of Ahlfeld and Mizuo, it will be seen that the others closely resemble one another, though differing in the complexity of their constituents. They are also exactly similar to the badly named dermoids of the ovary, which are in reality true teratomata, since they usually contain, in addition to epiblastic and mesoblastic structures, derivatives of the hypoblast, such as respiratory and intestinal mucous membrane, etc.* Similar tumours are not infrequent in the testicle, and the well-known congenital sacral teratomata are of the same kind; they are also sometimes found in the subcutaneous tissues, mediastinum, pelvic connective tissue, within the skull, etc.

Various hypotheses have been advanced to account for these tumours. According to one, they arise by parthenogenesis; an ovum, without having been impregnated, takes to growing and dividing on its own account; lacking its spermatie complement, its development cannot result in a perfect individual, but only in a confused jumble of tissues and malformed organs. This explanation might be true for teratomata of the ovary, or even, substituting "sperm cell" for "ovum," for those of the testicle, but it obviously cannot apply to teratomata, such as the orbital and sacral, which grow in situations where there are no germinal cells; and since all these tumours are exactly the same in structure, no hypothesis can be accepted which fails to include every form.

Another hypothesis derives the tumours from the impreg-

* A distinction is sometimes drawn between "dermoids" and "teratomata" of the ovary. The former are frequently cystic, and there is often some attempt at the arrangement of the tissues in systems—a piece of skin occurring in one place, a piece of intestine in another, etc.; the teratomata are solid, and the tissue of which they are composed—epidermis, hairs, sebaceous, sweat, and other glands, tubes and cysts lined with ciliated epithelium glands, connective tissues, etc.—are confusedly mixed. The tissues in a teratoma are ill-developed and embryonal in type, while those of a dermoid are fully developed, and have the appearance of being of the same age as the tissues of the host. It is evident that these differences represent types of the same tumour and are not fundamental: both are true teratomata according to the above definition. Most of the teratomata of the orbit have more resembled ovarian "dermoids" than ovarian "teratomata."

nation of polar globules. Before an ovum becomes impregnated, and indeed whether it becomes impregnated or not, two portions of its nuclear protoplasm are thrown off; inasmuch as these are part of the actual living substance which is to form the individual, it has been thought that, if they met with a spermatozoon, one or both of them, as well as the proper nucleus, might possibly become impregnated, a process which is said to have been observed in invertebrate ova; in that case the ovum, segmenting and growing to form an individual, would harbour within itself a second impregnated germ, capable of growth and of the formation at least of a formless conglomeration of tissues. While it is difficult to formulate any absolute disproof of this hypothesis, it must be regarded as very doubtful whether such a destiny can be ascribed to the polar globules. So far from their being liable to impregnation as a general rule, it seems that the germinal vesicle cannot fuse with the sperm cell until it has got rid of them. The polar globules never exceed three, yet a larger number of teratomata than this may be found in an ovary.

A third hypothesis has the advantage of including within its scope a large variety of abnormalities. It may be stated as follows. The two cells into which an impregnated ovum first divides presumably contain within themselves the potential representation of all three embryonic layers, and of all the tissues of the body—they are “totipotent.” Sometimes this is made manifest by their subsequent development, not into the tissues of a single individual but into two separate individuals; in which case twins will be produced who are alike in sex, and who closely resemble one another in bodily features. It is evident also that if one of these cells had been suppressed, the other would still have been capable of forming an individual.

As the ovum goes on dividing, the cells (blastomeres) of which it is composed gradually lose their totipotency, according to the well-known principle of Herbert Spencer that development is a progression from incoherent indefinite homogeneity to coherent definite heterogeneity, or, in other words, that

increasing specialisation goes step by step with increasing differentiation. Yet in the earliest stages, before the three blastodermic layers are formed, it is probable that the blastomeres are capable of developing into tissues derived from any or all of these layers, and that they have a certain power of compensatory adaptation, whereby, if some of their number are suppressed or set aside in development, the remainder are capable of replacing them, and of developing, in spite of their loss, into a perfect individual. After the formation of the blastodermic layers the potency of the cells is much limited, epiblastic cells, for instance, being incapable of developing into any tissue other than epiblastic, and similarly with the cells of the meso- and hypo-blast; at the same time the compensatory power of the cells undergoes limitation in the same sense, suppressed epiblast being replaceable by epiblast, but not by mesoblast, etc.

Applying these considerations to teratology, it has already been stated that the highest degree of differentiation of which a single ovum is capable is into two separate, equal individuals. Or one individual may grow at the expense of the other although they remain separate. Coming a step lower, the two individuals may be complete but joined together by a more or less extensive adhesion, as in the case of the Siamese twins. Or the placenta of one individual may acquire an adhesion, not to the maternal uterus but to the other individual; in this case the adherent foetus will derive its nourishment, not directly from the maternal blood but only indirectly, and through the partially venous blood of the other foetus; the parasite will lag behind in development, and more especially so if the adhesion be to some part of the second foetus remote from the main vascular organs and channels. This degree of parasitism is exactly illustrated by Mizuo's case (p. 81), in which the umbilical cord of the parasite was inserted into the orbit of the autosite; sometimes the attachment is to the mouth or elsewhere.

The division of the ovum to form two individuals may

also be partial, giving the various grades of aberanial or abcaudal fission.

In a yet lower rank come the cases in which the parasite, though still recognisable as a separate individual, begins to become fused with the autosite. This is illustrated in the case of Ahlfeld (p. 78), in which the buttock and leg of a second individual projected from the orbit. The abnormality is of the greatest rarity in the orbit, but it is not so uncommon to find the parasite attached to the roof of the mouth (*epignathus*), the front of the abdomen, the sacrum, and elsewhere; many examples will be found collected in the monograph of Ahlfeld.

The next stage is that in which no externally distinguishable limbs are formed, but only an amorphous mass, which contains, however, representatives of all, or most, of the tissues of the body, and even recognisable loops of intestine, long bones, etc. In this group come the cases of Lawson, Bröer and Weigert, Courant, v. Hippel, and the present case; as well as the ovarian and testicular teratomata, the sacral tumours, etc.

It is probable that all the degrees so far enumerated arise from the setting aside of blastomeres in the earliest stages of embryonic life, before the formation of the epi-, meso-, and hypo-blast; the autosite replaces the lost blastomeres and goes on growing to form a complete individual, while at some later stage, or perhaps from the first, the segregated blastomeres take on independent growth. The earlier the separation took place the greater, presumably, is the power of the isolated cells to form a second individual, so that the origin of the amorphous teratomata must be referred to a period relatively late, yet earlier than the full differentiation of the epi-, meso-, and hypo-blast.

After the differentiation of these layers the cells have no longer the power of producing complex neoplasms, but they may still be set aside, and if they subsequently take on growth will produce independent tumours. Thus isolated cells of the epiblast may produce brain-containing tumours

such as are mentioned on p. 82; or they may give rise to dermoids, and, according to the Cohnheim theory, they may also be responsible for the later development of rodent ulcers, epitheliomata, etc.: cells of the mesoblast may produce chondromata, osteomata, congenital kidney tumours, etc., and possibly also the whole group of the sarcomata: cells of the hypoblasts may produce certain congenital cysts, cysts lined with ciliated epithelium, as in the case of Wagenmann (p. 83), and perhaps the hypoblastic carcinomata.

It would seem also that the period of latency may vary greatly, and it is probably a general rule that the earlier the cells are segregated the less are they likely to remain latent; for instance, a tumour externally recognisable as a second individual never commences to grow after birth, but an amorphous teratoma may, at least in the ovary, and the sarcomata and carcinomata practically always do. Against the general applicability of this law, however, is to be set the fact that the orbital teratomata, though they may be fully as complicated as the ovarian, are always present at birth, while the ovarian often commence to grow only in later life (v. Hippel).

With regard to the abnormalities here discussed, from twins down to amorphous teratomata, there is another possible explanation of their origin. Twins are not always the result of the independent growth of two blastomeres of a single ovum: they sometimes originate in the impregnation and growth of two separate ova, in which case they may be of different sex, and without resemblance to one another. It is conceivable that one such impregnated germ cell might become adherent to, or included in, another, and might produce a more or less differentiated individual in the manner already described. An ingenious attempt to put the matter to the proof of experiment was carried out by Petrow (*Centralbl. f. allgem. Path. u. path. Anat.*, vol. xvii, p. 353, 1906). He made an emulsion of very early guinea-pig embryos, and injected it into the testicle of an adult animal. A tumour as large as the testicle itself had formed

in four weeks, after which it ceased to grow. Microscopically it contained brain tissue, epidermal cysts, cartilage, bone, muscle, and cysts lined with cylindrical epithelium, and furnished with glands. A smaller similar tumour also formed in the track of injection. V. Hippel repeated this experiment in the orbit, and obtained a small episcleral teratoma of practically identical structure. These experiments only show that the embryonal tissues of one individual may form independent tumours in the adult tissues of another. They do not imitate the actual conditions of early foetal life, and cannot be said to throw much light on the question whether the similar tumours now under discussion are the fruit of an independently impregnated ovum or of the same germ cell which produces the autosite. It seems most probable that the second of these explanations is correct; for the two individuals which result from a single ovum are developed within a single amnion, and are more likely to adhere or become included one in the other than two independent embryos. In the case of the deep seated amorphous tumours it becomes still more likely that they arise from cells of the autosite rather than from an independent impregnation. It is extremely improbable that two independently impregnated ova should ever come together at all in the wide extent of the genital mucous membrane. Moreover, where the sex of the parasite is distinguishable it is always the same as that of the autosite, and this, as already mentioned, is a characteristic feature of the twins which arise from the same ovum. If Holmes's case were not so doubtful on other grounds it would be of great interest to note the fact that the autosite itself was a twin; unless the extremely improbable supposition were entertained that three impregnations had taken place—one for each twin and one for the tumour—this would amount to proof that the tumour had developed from the same ovum as the autosite, not from an independent impregnation.

It would seem, therefore, that the tumour here described originated in a very early period of intrauterine life, by the

segregation of certain cells of the developing embryo, which subsequently took on independent growth. When did this independent growth commence? From the fact that the development of the globe and optic nerve was not interfered with, one may perhaps infer that it was relatively late in foetal life, when the formation of the eye was fairly advanced. It is known from post-natal observation, and might indeed be expected from their structure, that these tumours may grow with great rapidity; hence their considerable size at birth is not inconsistent with a late commencement of activity.

THE CHOROIDAL BLOOD SUPPLY OF THE RETINA.

By MALCOLM L. HEPBURN.

THE blood supply of the posterior layers of the retina comprising the pigment epithelium and the rods and cones is derived from the choroidal circulation, and under normal conditions the retinal vessels do not participate at all in the nourishment of the neuro-epithelium. There are, however, reasons for supposing that if the choroidal circulation should happen to be prevented from carrying on the usual blood supply, its place may be taken by the vessels from the retinal system (see papers in T.O.S. vol. xxviii, and R.L.O.H. Reports, vol. xvii). In the same papers I had occasion to remark on the definite order in which the different parts of the retina lose their function in cases of primary pigmentary degeneration, and showed that the affected areas do not follow the lines of direct contiguity, but that some process of selection is indicated; so that, when the field of vision shows extensive scotomatous areas, a few isolated patches of functional retina are still to be found in certain places, some of which occupy fairly constant positions on the charts.

After an examination of 50 cases of primary and secondary degeneration, I am convinced that this special feature is no mere accident, since it is of too frequent occurrence to be dismissed in this manner.

That certain parts of the retina show less inclination than others to resist the degenerative processes, I have observed very often, so that one sees time after time the order in which loss of function proceeds following the same general type. Thus, for example, in examining fields of vision in retinitis pigmentosa which have arrived at the stage when only central vision apparently remains, by varying the illumination or the size of the test object, I have often succeeded in eliciting a small area of visual acuity

downwards and outwards (corresponding to the upper and inner portion of the retina), and from this I conclude that this particular area is the one which immediately precedes the macula in order of degeneration (see Figs. 3, 4, and 5).

Not only does the retina degenerate in sections, but a certain class of case is met with where the degeneration tends to occur in localised areas, and to remain confined to the one particular region without showing any disposition to advance in any direction. I have come across one or two such cases in the course of my investigations, one of which I have already shown (R.L.O.H. Reports and T.O.S.), while two others are described in the present paper.

There are two possible explanations of this tendency to sectorial degeneration; either, there must be some inherent property belonging to the neuro-epithelium in the different portions of the retina rendering some parts more susceptible than others to the development of degenerative processes (Ginsberg); or, the blood supply of the neuro-epithelium of the retina is divided into a series of terminal vascular systems, the individual branches of which show varying degrees of liability to disease or obstruction, the extent of the damage being dependent upon the size or number of vessels involved. Of these two explanations, I regard the latter as the more probable, chiefly because of the occurrence of localised degenerative changes remaining confined to one special area for so long a time without undergoing any change.

Leber's work on the choroidal circulation is too well known to need repetition here, but amongst other points he drew attention to the fact that the choroidal vessels give off no branches, and only anastomose with others in certain places, *e.g.*, near the disc and in the most anterior part of the eye; and he also observed that the calibre of the individual capillaries varies in different places, being widest at the macula. Consequently, we should expect the clinical evidence to coincide with this anatomical arrangement.

If then we admit that the cause of the loss of the function

lies in the circulatory apparatus, it is at once apparent, from an inspection of a large number of visual fields, that we can divide the blood supply of the posterior layers of the retina into three main sections ; viz., the macula, the mid-periphery, and the extreme periphery. These divisions apply only to the parts of the retina which are visible ophthalmoscopically, probably not much further than the equator, since beyond what we now call the extreme periphery is an area which is also supplied by the choroidal circulation (the recurrent branches of the anterior ciliary), but which does not come into the present discussion. Just as the nerve fibres of the retina are arranged in three main divisions which occupy definite positions in the optic nerve, so I think we can show that the choroidal circulation is probably arranged on the same lines, and I propose to approach the subject from this point of view.

The Macula.

There is plenty of evidence to indicate a separate blood supply for this part of the retina. One has only to mention the cases of hæmorrhage into the macula, senile degenerative changes of all degrees, etc., all of which are limited very precisely to the macular region, and exhibit a strongly localised tendency. It is here, however, that there is the greatest resistance to degenerative processes which attack the retina as a whole, this being the last part to yield in cases of primary pigmentary degeneration.

It is not unknown for the central vision to be lost while there is yet some peripheral acuity still remaining, and I published a case of this nature in my paper in the T.O.S., but it is distinctly rare, and when it occurs there has always been a history of a specific taint leading to the irregularities which are so characteristic of this condition.

When once the macula has become involved the chances of ultimate recovery are more remote than in disease of any other part of the retina. The reason for this is,

I think, to be found in the fact that the retinal vessels in this region are practically non-existent, being of such a minute character that there is very little chance of collateral circulation from this source for purposes of restoration of vitality. The macular region may be said to extend seldom beyond the 20° circle on the visual chart as regards the vascular supply.

The Mid-Periphery.

This region comprises rather a wide area on the visual chart, and extends from about the 20° circle to the 60° on the temporal side, and 40° circle on the nasal side.

As I have mentioned elsewhere (T.O.S., vol. xxviii), it is this portion of the retina which manifests the earliest sign of failure of vision in cases of primary pigmentary degeneration, and is evident clinically as the ring scotoma which may be either complete or partial, absolute or relative, according to the extent of interference with the blood supply.

Apparently it is not yet the opinion of all observers (Shoemaker, etc.) that the ring scotoma is present in every case of primary retinal degeneration, but I have never yet experienced any difficulty in finding it, though occasionally only in a modified form (Fig. 1). True, it does not occupy exactly the same position in every case, but weak spots are present in certain places where the examination must be conducted with special care in order to detect the scotoma.

The part of the mid-periphery which shows this special weakness must be looked for generally at the upper and inner portion of the retina, giving rise to a scotomatous area down and out in the visual field, and it is here that extra care is necessary in making the examination. Extension generally takes place both circumferentially and meridionally, starting from this point.

Although usually affection of this region is the precursor of a general degeneration, instances have occurred where a

localised pigmentary degeneration has remained confined to one part of the circumference without showing any disposition to extend at all in any direction, even after the lapse of some years. One such case has already been described by me in the R.L.O.H. Reports (Case 6), and in the present paper Fig. 2 represents the field of vision taken in the same case after an interval of two years, and a certain amount of improvement is evident in the right eye, while in the left there has been no extension in the mid-peripheral region, but the extreme periphery in the area corresponding to the scotoma has become involved.

Another case published by me in the same paper (Case 2) shows the charts of fields of vision, the second one taken after a period of two years, and I commented on the changes which had occurred in the interval.

And this brings us to the next point, which is, that not only is the mid-peripheral region the place where some amount of recovery is evident after a certain time, but also it is the part where one notices the greatest variation between the ophthalmoscopic appearances and the defects in the visual fields, very gross changes in the choroid with excessive pigmentary deposits being often consistent with a remarkably good field of vision. I have shown charts of many such cases in my paper in the T.O.S., vol. xxviii, and I have met with several others of a similar nature since.

The reason for this seems to me to lie in the condition of the retinal circulation in this particular part of the fundus. The vessels are here of larger dimensions than elsewhere, thus facilitating the formation of a collateral circulation from this source when the blood supply from the choroidal system is interfered with through obstruction of the normal channels. The more detailed distribution of the blood vessels for this part of the retina presents greater difficulties than in the other regions. It might appear from the very sharply limited scotoma in Case 6 in R.L.O.H. Reports, vol. xvii, that an upper and a lower

branch contribute the blood supply of the mid-periphery, and possibly this is the case; but in some of the fields there are indications that each main trunk divides into several branches, each of which represents a miniature terminal vascular system.

The Extreme Periphery.

By this term I mean the most peripheral part of the retina which is visible with the ophthalmoscope, and which roughly comprises that part of the visual field lying beyond the external limits of the mid-periphery, viz. beyond the 60° circle on the temporal and the 40° circle on the nasal side. Anatomically, it corresponds to about the equator of the eyeball.

I am inclined to question the value attaching to the anastomosis of the posterior ciliary arteries with the recurrent ciliary branches, and doubt whether this communication takes place far enough back to be of any use for visual purposes.

In primary pigmentary degeneration the extreme periphery comes next in order to the mid-periphery as regards loss of function, and shows considerable irregularity in the susceptibility of its different parts. Speaking generally, one may say that the temporal side of the retina is more commonly affected than the nasal, either in whole or in part, and often remains as a permanent defect while the rest of the retina escapes. On the other hand, I have observed several times (Figs. 27, 28, 30, T.O.S.) that a small portion of the retina down and out, corresponding to up and in on the chart, often holds out in primary degeneration until only the macula and part of the nasal side remain functional.

Again, the upper half of the retina is more resistant to the degenerative processes than the lower, so that in looking over a large number of charts one notices that the scotoma is present most frequently in the upper part of

the visual field rather than the lower; but the part of the extreme peripheral region which resists longer than any other, with the exception of the macula, is a small area upwards and inwards (Figs. 3, 4, 5), and this fact I have repeatedly demonstrated in the course of my examinations. It is one of the most constant factors in all cases of disease associated with loss of function of the retina, and Hancock has drawn attention to it in cases of embolism of the central artery of the retina (R.L.O.H. Reports, vol. xvii, Plate 3, p. 428).

It is also remarkable that, whereas the nasal side of the extreme periphery upwards and inwards is the part where function remains longest, the corresponding area in the mid-periphery is the spot where one looks for the earliest sign of visual defect (Figs. 1, and 44, 45, T.O.S.).

I have come across one case (Fig. 6) where almost the whole of the nasal half of the extreme periphery showed the typical ophthalmoscopic appearance of primary pigmentary degeneration, and there was also present a small mass of white fibrous tissue projecting forwards at the nasal margin of the disc. It seems only natural to connect the two things together, and to conclude that the main trunk supplying this quadrant of the extreme periphery was obstructed at this point, indicating that the blood supply of this portion of the retina is derived from a branch of a posterior ciliary artery given off far back close to the entrance of the optic nerve, which passes forwards to its destination without contributing to the mid-periphery on the way.

Another case of a similar nature, though perhaps not so striking, is seen in Fig. 7. Here the scotoma involves the whole of the extreme periphery down and out, though some of the mid-periphery is also affected; and the deep choroidal disturbance was seen ophthalmoscopically to be situated close up to the margin of the disc in a corresponding situation. When once the retina has lost its function in this region, there is very little chance of recovery owing to

the size of the retinal vessels, which alone can be relied upon for a collateral blood supply.

The subdivision of the main trunks and their distribution can be inferred from a study of the visual fields and their defects. Fig. 6 shows the extent of damage in the case of the blocking of a vessel near the disc; and the question suggests itself as to how many of these vessels are given off in this situation for the supply to the extreme peripheral regions. There is some reason for saying that there must be about eight vessels supplying the most anterior functional part of the retina, since the blocking of a large vessel containing many branches on the nasal side would have led to disturbance of function in a far larger area than we see in the figure. (Compare Figs. 6, 7, 8, and 9.)

I have discussed this subject from a purely clinical standpoint, and at present detailed anatomical proof is entirely wanting. At the same time, Leber's researches led him to conclude that the larger posterior ciliary arteries have little or no communication with one another; and Coats, in his paper on Posterior Scleritis, discusses the subject very ably in his "remarks," and is forced by his pathological and histological observations to adopt the same attitude, and he agrees with Leber in throwing doubt on the apparently intimate anastomosis of the posterior choroidal circulation. On my side, I submit that the loss of function, as evidenced by the presence of specially situated scotomata in the field of vision, represents the interference with the normal source of nutrition carried out under ordinary circumstances by the choroidal circulation, and the fact that this loss of function can remain limited for years to one particular part, without showing any sign of extension, indicates that a single vascular system can be involved apart from any of the others.

Again, the actual changes visible with the ophthalmoscope give additional clinical support to this view of the choroidal blood supply of the retina, and present a different picture according to the size of the vessel or vessels involved. For example, we are all familiar with a well-defined

tubercular choroidal inflammation occupying the macular region, and the whole affection, together with all the subsequent changes, are strictly limited to this area from beginning to end. This points to the blocking of a large vessel by a tubercular metastatic deposit. On the other hand, the more superficial changes occupying the same localised area, seen as senile degeneration, Tay's choroiditis, and slight pigmentary disturbance, represent a milder condition, affecting the capillaries of the same circulatory system. The same may be said of the mid-peripheral region; well-defined localised areas of inflammation, with all the signs of choroidal disturbance and pigmentary proliferation, are sometimes found here occupying different positions, and in other cases the patchy variety is the more prominent feature, giving rise to irregularly-shaped areas of atrophy with deposits of pigment in the neighbourhood.

I do not mean to convey the impression that all choroidal changes are the result of obstruction in some branch of the posterior ciliary vessels, since inflammatory deposits also occur in the stroma leading subsequently to small areas where the retina and choroid become adherent. At these places there is necessarily loss of function, which becomes apparent in the fields of vision as small scotomata. I should say that it is not possible to differentiate ophthalmoscopically between this variety and that caused by the blocking of a small vessel. Primary pigmentary degeneration of the retina represents a general disease commencing in the terminal capillaries, and spreading gradually to the larger vessels.

So far, it is comparatively easy to formulate a theory of separate terminal circulatory systems, but it is a different question when we come to enquire the reason why some of the sections show more susceptibility to obstruction or disease of their vessel walls than others.

No doubt the blocking by an embolus of any single branch may be a matter of accident, but this cannot apply to cases of thrombosis or disease of the coats of the vessels. As far as one can see, there is nothing in the position, size of

the vessels, or condition of the blood current that would render them specially liable to disease, and at present I am at a loss to understand the reason for the peculiar susceptibility of this particular vascular system. I incline to the belief that the class of cases which we have been discussing is in its nature allied to the different varieties of Reynaud's disease. There are several stages of this complaint which may either remain stationary or progress, and undoubtedly the cause is a vascular one, though so far the pathology is in a state of uncertainty.

Lightly shaded areas = relative scotoma.

Darkly shaded areas = absolute scotoma.

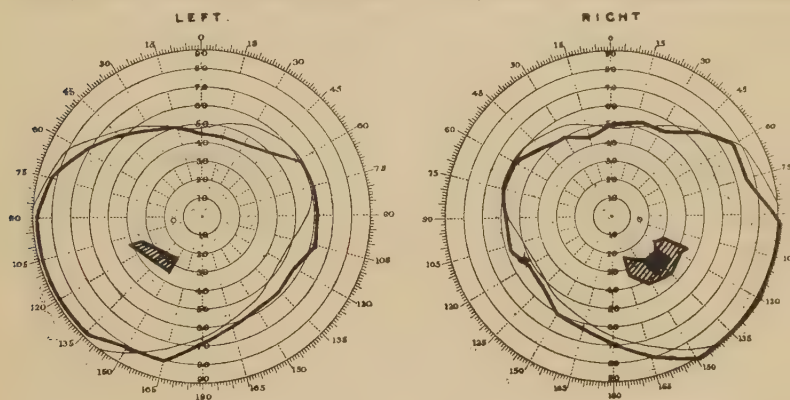


FIG. 1.

Alex. O., aged 33, under the care of Mr. Hancock at the Central London Ophthalmic Hospital, suffering from Retinitis Pigmentosa.

Night blindness as long as he can remember.

Hands show signs of weak circulation.

No specific history.

R. and L.V. 6/5 in each eye with - 0.5.

Typical pigmentary changes in both fundi, with long lines of pigment round the veins on the nasal side. Other parts of the fundi free, but the mid-periphery has a somewhat grey appearance.

Discs only a little indistinct on the nasal side.

Retinal vessels reduced in size.

These fields are taken from the same man figured as Case 6 in the R.L.O.H. Reports, vol. xvii, after two years interval.

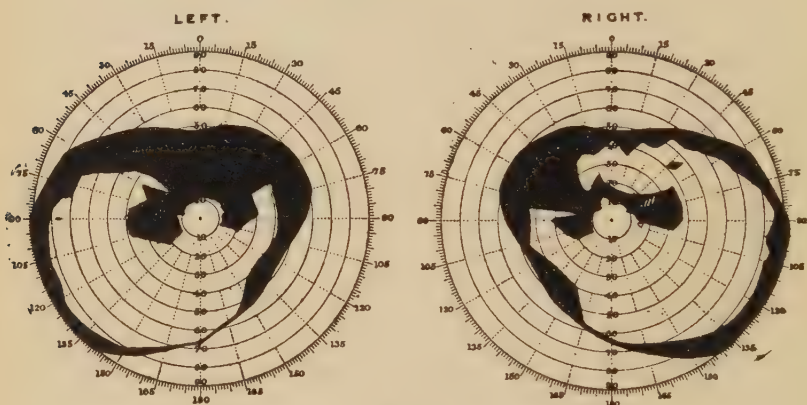


FIG. 2.

He had frequently complained of pain at the back of the left eye during this time.

Job A., aged 40, under the care of Mr. Lawford at Moorfields, suffering from Retinitis Pigmentosa.

Night blindness ever since he can remember.

Cold hands and feet *not* marked.

R. and L.V. 6/18.

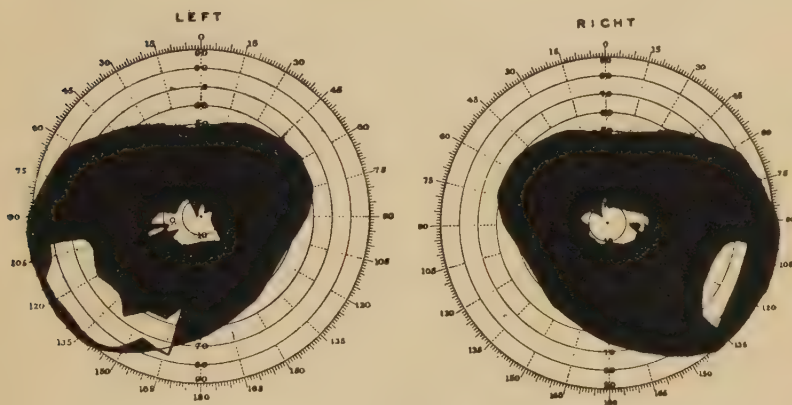


FIG. 3.

Small posterior lens opacities in each eye.

Both fundi show typical pigmentary changes all over, which are most marked in the mid-periphery.

Discs waxy and retinal vessels reduced in size.

Albert R., aged 43, under the care of Mr. Gunn at Moorfields,
suffering from Retinitis Pigmentosa.

Night blindness six or seven years.

Cold hands and feet to a certain extent.

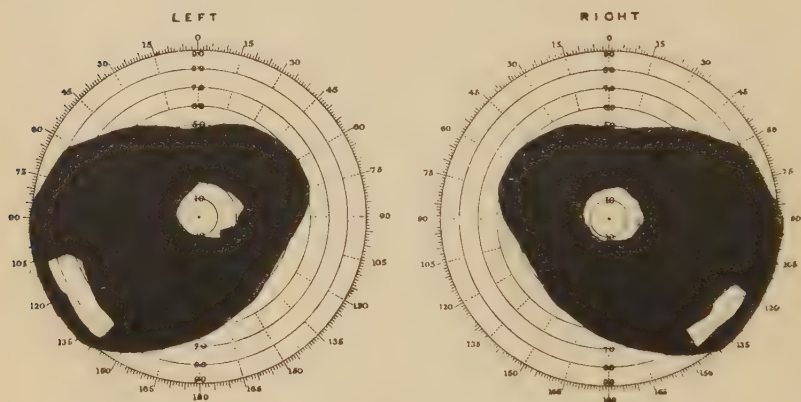


FIG. 4.

No specific history.

Posterior cortical cataract and some vitreous opacities.

No atrophic areas in the choroid and very little pigmentation,
but what there is combines the blotchy type with some typical
degenerative change.

Benjamin P., aged 38, under the care of Mr. Lawford at Moorfields, suffering from Retinitis Pigmentosa.

Night blindness, 15 years at least.

Cold hands and feet, with feeble circulation.

No specific history.

R. and L.V. 6/24 pt.

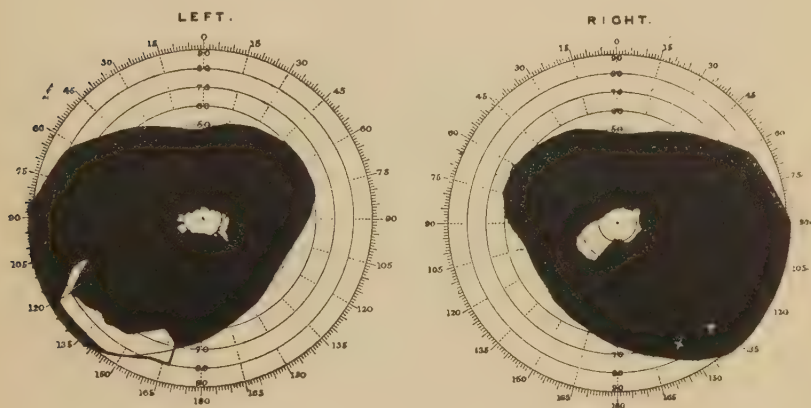


FIG. 5.

Vitreous opacities in both eyes and posterior polar opacities in the lenses.

Both fundi show much the same changes, viz., typical pigmentation with some smaller clumps, most marked in the mid-periphery.

Dises waxy and retinal vessels reduced in size.

Note $\times \dots \times$.—The portion of the right field between the two crosses is only functional to brilliant illumination.

Julia B., aged 14, under the care of Mr. Parsons at Moorfields.

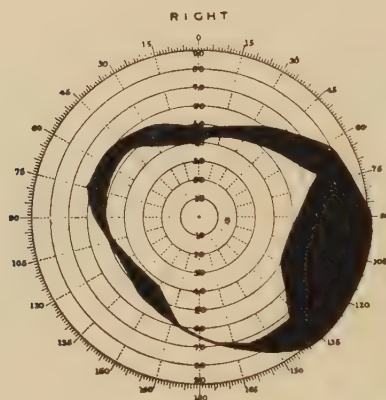


FIG. 6.

There was no history and the retinal condition was discovered accidentally.

R.V. + 3 cyl. $\downarrow$ = 1/60.

L.V. 6/12 c - 0.5 = 6/6.

Geo. M., aged 26, under the care of Mr. Treacher Collins at Moorfields, on December 16, 1907.

There was a somewhat indefinite history of being struck on the left eye with a piece of wood.

A faint grey opacity was discovered on the cornea.

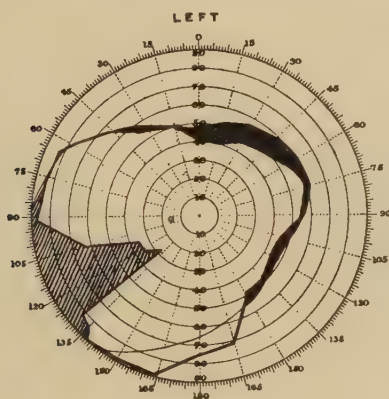


FIG. 7.

On December 23, 1907, a note is made as to the presence of K.P. and a white œdematous mass on the inner side of the disc.

R.V. 6/6.

L.V. 6/36.

On March 12, 1908, the vision was 6/6, and an area of deep choroidal disturbance was present on inner side of the disc.

James S., aged 52, under the care of Mr. Holmes-Spicer at St. Bartholomew's Hospital, suffering from Retinitis Pigmentosa. Night blindness, four years. Cold hands and feet. No specific history.

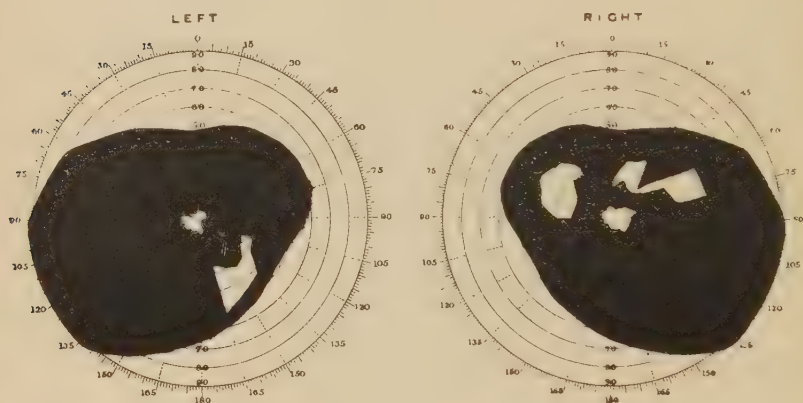


FIG. 8.

Both fundi show typical pigmentary changes all over the extreme periphery. Faintly granular appearance of mid-periphery; with coarser pigmentation further out which follows the course of the vessels. Choroid grey in extreme periphery, but no atrophic areas.

Disc rather waxy and retinal vessels reduced in size.

Thomas S., aged 48, under my care at The Hampstead General Hospital, suffering from specific retino-choroiditis.

Night blindness.

Never cold hands and feet.

Specific history 12 years ago.

Old iritic adhesions, but no vitreous opacities.

Lenses clear.

R.V. Hand movements only.

L.V. 6/12 pt. with correction.

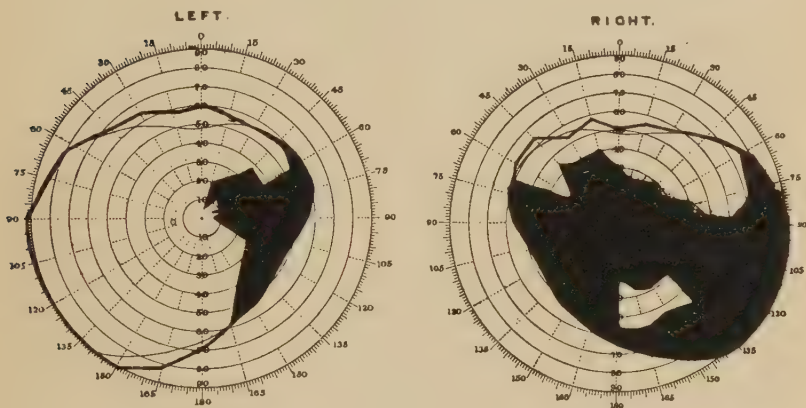


FIG. 9.

Greyish fundi in both; very marked pigmentation on temporal side where it is of a blotchy type. Typical degenerative changes on nasal side up and in. Right side worse than left. R. disc waxy and retinal vessels reduced in size. Left disc edge blurred and rather paler than normal.

Conclusions.

1. The posterior layers of the retina are supplied entirely by the posterior ciliary arteries, which under normal conditions, receive no assistance from any other vascular system.
2. The posterior ciliary arteries distribute their blood

supply in three main sections, which nourish separate parts of the retina.

3. These three main sections supply :—

- (a) The macular region.
- (b) The mid-periphery.
- (c) The extreme periphery.

Each one is distinct from the others, constituting individually a series of terminal circulatory systems and showing varying degrees of susceptibility to disease.

4. The division into these three areas is a more or less arbitrary one, since there is no accurate demarcation in the field of vision corresponding to the point where one begins and the adjoining one ends ; but it may be roughly stated that the macular region seldom extends beyond the 20° circle, and the mid-periphery, although subject to slight variations, seldom extends beyond the 60° circle on the temporal side, and 40° on the nasal side.

5. Each main vascular area is again subdivided into several regions, each of which shows the same characteristics as the main area, and exhibits a similar variation in susceptibility to disease.

6. In primary pigmentary degeneration the whole system is involved ; and under these circumstances the degenerative processes are in a state of progression and follow a definite order, the mid-periphery being the first to succumb and the macula the last of all.

7. In secondary degeneration, any one of the three may be affected either in whole or in part, and many irregularities are shown, as well as the possibility of the affection remaining isolated to one part, often without giving any sign of extension after a number of years.

8. As regards recovery, after the retina has once lost its function in any part, this depends entirely on the possibility of establishing a collateral circulation through the retinal system. The mid-periphery shows the greatest response to any attempt at restoration of vitality, whilst

the macula, although it resists disease longer than any of the others, is generally entirely beyond repair when once the blood supply through the choroidal circulation has been interfered with. Therefore, vascular affections of the macula are attended with a grave prognosis, while the mid-periphery frequently contains gross pathological changes with but little disturbance of function.

9. The probable reason for the behaviour of the different parts towards recovery is to be found in the extremely poor retinal circulation in the macular region, which does not lend itself readily to a collateral circulation, whereas in the mid-periphery the retinal vessels are at their maximum size, thus more easily encouraging the establishment of a new blood supply.

10. One is justified in drawing the foregoing conclusions from a critical examination of the fields of vision, and they support Leber's contention that the posterior choroidal vessels anastomose very little with each other. There is also evidence to show that the branches supplying the extreme periphery are given off close to the entrance of the optic nerve.

I have to express my gratitude to Mr. Gunn, Mr. Lawford, Mr. Treacher Collins, Mr. Holmes Spicer, Mr. Parsons, and Mr. Hancock, for their kindness in allowing me to make use of their cases for the purposes of this paper.

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A CLINICAL STUDY OF POSTERIOR TRAUMATIC CATARACT.

By A. C. HUDSON.

THE clinical study of the behaviour of the crystalline lens under the influence of injury, whether direct or indirect, is one which presents on the one hand exceptional interest, and on the other peculiar opportunities, owing to the fact that the observer is here dealing with one of the very few tissues of the body which are under normal conditions transparent. This study is, however, one which is very apt to be interfered with, or at least rendered difficult, by the rapidity of onset and development of changes resulting in opacification of the observed tissue, either in part or in whole, and, in the case of the human subject, by the effects of associated injury of neighbouring tissues and by personal considerations which may be contra-indicatory to frequent and exhaustive examination. It is probably as the result of the above considerations that little has hitherto been published descriptive of the precise mode of development of, and course pursued by, those striking changes which may so frequently be observed at the posterior part of the human lens as the result of injury.

The writer, having had the opportunity of studying a considerable number of cases illustrative of the formation and subsequent modification in appearance of such changes in the injured lens, hopes to be able, by means of a somewhat detailed description of the appearances observed, to add something to the present knowledge of their ætiology and significance. The conclusions arrived at will be supported by a description of certain changes occasionally observed at the anterior surface of a lens as a result of injury.

For permission to make use of the clinical notes of the following cases the writer is indebted to the courtesy of the members of the surgical staff of the Royal London Ophthalmic Hospital. The periods of time mentioned in

the notes refer in all cases to the interval of time which had elapsed since the receipt of injury.

GROUP 1.—*Case exhibiting Earliest Appearances of Posterior Traumatic Cataract.*

CASE 1.—Richard W. (Mr. Collins), æt. 17. High myopia.

5.12.07 (12 noon). L. Needling lens.

6 p.m. L. Some striate opacity at posterior aspect of lens in direction of lens fibres (Fig. 1). Opacity is limited to central region.



FIG. 1.

In this, as in all the other figures except the last, the appearances are those seen by transmitted light with a plus lens of not less than 20 D.

6.12.07. Condition as yesterday, but more extensive. Radial striation at margin of lens at points opposite to the feather-like striated figures which are seen lying in the position of the main lines of the posterior lens star.

8.12.07. Feathery opacity, mid-ribs of feathers corresponding to lines of posterior lens star, extends to margins of lens all round. Some of these main limbs bifurcate near their extremities; no vacuoles seen. By oblique illumination the opacity has a sheen like dense fibrous tissue, and appears to lie at the posterior surface of the lens. In certain lights the processes appear light on one side of the mid-rib and dark on the other.

9.12.07. Lens matter coming forward through rent in capsule.

Indefinite anterior opacity obscures posterior opacity.

Lens became broken up.

GROUP 2.—*Cases exhibiting Sequence of Typical Changes in Posterior Traumatic Cataract in the Course of Evolution and Involution.*

CASE 2.—Morris D. (Mr. Gunn), æt. 10.

26.11.07. Congenital upward dislocation of both lenses.

R. Lens has been removed. V. = 6/9 unaided.

L. V. = 6/36 and J_i at 2 inches.

27.11.07. L. Needling lens.

Two days. L. Feathery posterior lens opacity (Fig. 2).



FIG. 2.



FIG. 3.



FIG. 4.

Three days. Eastern part of figure shows vacuolation of mid-ribs, with hair-like processes; one vacuolated process (Fig. 3).

Four days. The three main featherlike sections have increased in size so as to touch one another laterally; there are some vacuoles among the processes (Fig. 4).



FIG. 5.



FIG. 6.



FIG. 7.

Eleven days. The whole figure is composed, apparently, of vacuoles, but only some can be seen for certain; the remainder of the figure might be formed by linear opacities joined by curves. The angles of the figure, excepting the N.W. one, have become rounded off by the cutting off of their points, which are represented by isolated vacuoles.

Twelve days. Isolated vacuole S.E. gone.

Thirteen days. N.W. angle becoming rarefied; its extremity is represented by one isolated vacuole (Fig. 5).

Fifteen days. N.W. angle represented by discrete granules and vacuoles (Fig. 6). Central S. vacuole gone.

Eighteen days. Only granules at N.W. angle. All outlying vacuoles gone.

Twenty days. Whole figure appears definitely vacuolar (Fig. 7).

Twenty-three days. W. segment of figure almost entirely granular; other segments of more open pattern.

Twenty-nine days. Posterior opacity has completely disappeared, except for some very fine discrete dusty dots. Anterior opacity is circumscribed with rather indefinite border.

CASE 3.—Morris D. (Mr. Gunn), æt. 10. (Same case as Case 2.)

28.12.07. L. Second needling of lens.

One day. L. Extensive feathery opacity, extending in many places to margin of lens, of similar character to that observed in Case 1. A few elongated vacuoles between marginal striæ below, and also amongst more central striæ.

Three days. In outer part of figure thickly outlined spaces in mid-rib, and more delicately outlined ones in contiguous part of figure elongated in direction of lens fibres; some hair-like extensions from these, also in the lines of the lens fibres.

Five days. Posterior opacity has a decidedly more open mesh-work, both in the mid-ribs and in their lateral extensions.

Six days. Process of figure represented yesterday by four contiguous vacuoles is now represented by discrete vacuoles of different size.

Ten days. Mesh-work of figure more open, but opacity more extensive down and out.

Twelve days. Opacity can be seen to pass round the lower border of the lens just at the surface.

Fifteen days. Mid-ribs becoming irregularly granular. Lateral extensions for the most part fairly clearly vacuolar, in some parts more granular. Intervening parts between main processes clear except for very fine striation.

Nineteen days. Marginal striated border is replaced by vacuoles.

Thirty days. Anterior opacity rather more extensive. Radial

creases in anterior lens capsule. Rarefied star posteriorly, much less extensive, almost entirely granular. Some discrete marginal vacuoles down and out.

Ninety-four days. Irregular granular posterior opacity in the form of a central star with broad stunted rays. Periphery quite clear.

As the result of subsequent operation the lens became opaque.

GROUP 3.—*Further Cases illustrative of the Conditions and Changes in the various Stages of Posterior Traumatic Cataract.*

CASE 4.—Sarah S. (Mr. Lawford), æt. 19.

Congenital downward dislocation of both lenses.

25.9.08. R. V. = 6/60 and J 8 at 3 inches. T + 1.

L. V. $\bar{c}$ - 6 Ds. = 6/18.

R. Lens needled three times ; no posterior opacity.

17.10.08. R. Needling of lens with Ziegler's knife.

Three days. R. Stellar posterior opacity, with nine rays, by oblique illumination appears grey, by transmitted light apparently composed of dark lines enclosing elongated and round spaces. When the patient looks downwards the extremities of the uppermost rays can be traced over the upper margin of the lens just on to the anterior aspect. They appear to lie just at the level of the lens surface, which shows no corresponding elevation. The grape-like fringe at the extremity of the upper rays can be seen when the patient looks down both by oblique and transmitted light ; it does not appear to be in direct contact with the anterior lens opacity. There is no separate marginal opacity.

Six days. To transmitted light the feathery linear formation in the rays is much less obvious, the vacuolar formation becoming predominant, especially in the lower and outer rays, where the vacuole-like components of the figure are larger than at the last note and to some extent discrete.

Thirteen days. Parts of figure composed of large and small discrete vacuoles, with some faint linear markings between them,

Lens became opaque after further operation.

CASE 5.—Clara B. (Mr. Gunn), æt. 20.

23.10.08. High myopia.

24.10.08. L. Needling lens.

26.10.08. L. Posterior lens opacity composed of fine lines and vacuoles radially disposed so as to form a star with about 20 rays.

28.10.08. L. Opacity covers whole posterior surface of lens and is vacuolar throughout, with thick lines between the vacuoles. Under oblique illumination the lines have an appearance as of fine, glistening, fibrous tissue.

29.10.08. L. Opacity does not completely involve the posterior surface of the lens, there being some clear spaces between the terminal parts of the rays. The linear markings are finer.

4.11.08. L. Posterior opacity, seen with difficulty, extends over the whole posterior surface of the lens. The linear markings are coarse and scanty, and enclose rather large irregular clear areas; the radial conformation can, however, still be traced.

Further observation impossible owing to increase of intervening opacity.

CASE 6.—Joseph S. (Mr. Morton), æt. 41.

3.9.08. L. Injured to-day by a piece of metal.

State on admission:—

L. Perforating corneal wound at inner limbus; hole in iris behind it. Feather-like striated lens opacity extending from, and striated marginal opacity on each side of, region of wound.

Skiagraphy shows no foreign body.

Three days. L. Rays of feather replaced by granules and one vacuole. Marginal striæ gone. V. $\bar{c} + 1$ Ds. = 6/18.

One month. L. Posterior granules fewer; vacuole has disappeared. V. = 6/6.

CASE 7.—Robert C. (Mr. Spicer), æt. 19.

16.8.07. Two days ago was struck in L. by a fragment while drilling iron.

Condition on admission:—

L. Lids œdematous; conjunctiva injected; a little mucopurulent discharge. Triradiate perforating corneal wound near

outer limbus, from which extends grey interstitial corneal striation. Iris inflamed; small hæmorrhages on its surface. Viscid hypopyon. Foreign body lies embedded in the lens, surrounded by lymph, which extends to the corneal wound. V. = 6/60.

17.8.07. Foreign body removed through wound of entry, and anterior chamber washed out with saline solution.

18.8.07. L. Aqueous clear; no hypopyon. Iris fairly bright.

2.9.07. L. Feathery posterior lens opacity. Grey anterior opacity. V. = 6/60.

Four months. L. Small, dense, grey localised anterior lens opacity. Posterior opacity less dense, composed of discrete vacuoles of varying size, with some intervening dots. Fundus normal. $V. \bar{c} \frac{+1 \text{ Ds.}}{+1 \text{ Dcyl. } 35^\circ \text{ down and in}} = 6/12.$

Five months. L. Posterior rarefied open-work opacity.

Six months. L. Posterior opacity is composed of vacuoles so small as to resemble granules; some rather larger discrete vacuoles in inner part.

One year. L. Posterior opacity granular.

Two years and three months. L. Posterior opacity granular; some very dense granules, grey to oblique light; almost no vacuoles, except one or two behind lens scar. V. = 6/18.

$\bar{c} \frac{+0.75 \text{ Ds.}}{+0.75 \text{ Dcyl. } 25^\circ \text{ down and in}} = 6/12,$ and Ji with difficulty.

CASE 8.—Henry M. (Mr. Lawson), æt. 23.

24.8.08. R. Injured five days ago by a fragment of steel.

State on admission:—

R. Perforating scar in lower part of cornea; hole in iris behind. Dense opacity in lower part of lens, and festoon of large vacuoles along its lower equator. Posterior stellar opacity, consisting of leaflets composed of fine lines, with appearance of some vacuoles in the inner lower leaflets near their extremities. V. = 6/12.

27.8.08. R. Foreign body removed by route of entry.

Sixteen days. R. Posterior opacity becoming granular. Festoon below not seen. $V. \bar{c} + 1.5 \text{ Ds.} = 6/12.$

Thirty-three days. R. Posterior opacity almost entirely vacuolar and granular. V. $\bar{c} + 2$ Ds. = 6/9.

Six weeks. R. Small detachment of retina. V. $\bar{c} + 2$ Ds. = 6/12.

Eight weeks. R. Lens opacity less. Universal detachment of retina. V. = hand movements.

Fifteen months. R. Posterior opacity consists almost entirely of granules ordinated to some extent in the lines of the lens fibres; some discrete vacuoles. Some grey striation in line of fibres visible by oblique illumination.

CASE 9.—Thos. B. (Mr. Marshall), æt. 20.

2.11.06. L. Injured by a thorn five weeks ago; sight interfered with at once, and became gradually worse.

Condition at first attendance:—

L. Perforating scar down and out, just outside limbus. Some anterior lens opacity in region of wound. Central posterior opacity of dense lacework type. V. = 1/60.

Sight began to improve about one month later.

Two months. L. V. = 6/60.

Two years. L. Small dense anterior opacity. Posterior lens opacity down and out, which does not quite reach to the centre, and is composed of dusty fine granules with three small vacuoles and some rather dark striation. V. = 6/9, $\bar{c} - .75$ Dcyl. 30° down and out = 6/6. R. V. = 6/24, $\bar{c} - 1.75$ Dcyl. 30° down and in = 6/6 partly.

Three years. L. Posterior opacity is less in extent, and is composed of granules with two very minute central vacuoles and some very delicate striation. V. = 6/6, made worse by cylinders or plus lenses. R. V. $\bar{c} - 1.75$ Dcyl. 30° down and in = 6/6.

CASE 10.—Wm. C. (Mr. Lawson), æt. 22.

27.6.08. L. Struck by a file 17 days ago.

State on admission:—

L. Central corneal scar. Posterior lens opacity in the form of a thick horse-shoe with a small opening above. Opacity appears to be composed of vacuoles, many of the marginal ones having fantastic shapes like hearts, rosettes, and splinters of glass

(Fig. 8, which represents lower inner border). Evidence of old choroido-retinitis and large detachment of retina.



FIG. 8.

One month. Vacuoles rather larger.

Three months. L. Posterior opacity is composed of large vacuoles and lobulated vacuoles. Many of the vacuoles are discrete.

Four and a-half months. L. Opacity is decidedly less dense, and is composed chiefly of large vacuoles, for the most part discrete. Some granules also to be seen.

Eighteen months. L. Posterior opacity is almost entirely granular; a very few small vacuoles to be seen. Behind inner part of opacity there is an appearance as of concentric folds at the posterior surface of the lens; well-marked parallax between these lines and opacity. The appearances were such as might be attributed to wrinkling of the posterior capsule dependent on diminution in the size of the lens.

CASE 11.—Wm. J. (Mr. Lawson), æt. 60.

24.9.08. Struck in R. eight or nine weeks ago while breaking stones.

State when first seen :—

R. Perforating scar at inner edge of cornea. Pupillary margin of iris lacerated and carried into lens. Very faint, dusty track through lens to hole in posterior capsule represented by a dark ring round a clear space. To the outer side of this ring are irregular radially-disposed wings of opacity of indefinite structure; above, and separated from it by some granular opacity, are two feather-like groups of striate opacity (Fig. 9). Foreign body in vitreous.

Fifteen weeks. R. Edges of hole in posterior capsule appear thicker and somewhat contracted; dark line in centre. Opacity

to outer side is less extensive, and appears condensed, presenting a resemblance to fine foam. Of the two previously striate opacities above, one is represented by an irregular open-work and the other by granules (Fig. 10).



FIG. 9.



FIG. 10.



FIG. 11.

Sixteen months. R. Hole in posterior capsule represented by fairly uniform opacity. Original outlying opacities have left no trace except for two or three isolated vacuoles and some granular opacity around scar, two processes of which obviously correspond with the original feathery opacities (Fig. 11).

CASE 12.—Arthur M. (Mr. Lawford), æt. 23.

25.5.08. L. Injured 13 days ago by a fragment of steel.

State on admission :—

L. Perforating corneal scar and hole in iris behind it. No anterior or interstitial lens opacity, but hole in posterior lens capsule with palisade of vacuoles above and along its outer side. Some fine granules at posterior surface of lens a short distance internal to the hole. Foreign body in vitreous.

$$V. \bar{c} \frac{+0.75 \text{ Ds.}}{+0.5 \text{ Dcyl. V.}} = 6/6.$$

27.5.08. Foreign body brought into anterior chamber and removed through corneal incision.

5.6.08. L. Posterior lens opacity has increased in its temporal part.

Five months. L. Wound in posterior capsule appears dark and contracted, and is surrounded by a rather dense granular band. The outlying opacities are faint, and entirely composed of discrete granules, except for one small collection of small vacuoles up and in. $V. \bar{c} = 6/9$ and $6/6$ partly.

CASE 13.—Henry E. (Mr. Gunn), æt. 41.

17.12.07. Was struck in L. by a flying fragment while breaking stones to-day.

State on admission :—

L. Central corneal wound. Lymph in pupil. Lens clear and not wounded. Skiagraphy shows no foreign body.

Eleventh day. L. Posterior polar opacity, in form of large flat vacuoles of angular outline (Fig. 12).

Fourteenth day. L. Main posterior figure as in Fig. 13.

Nineteenth day. L. Tail of largest figure is separated from the body by a dark line (Fig. 14).

FIG. 12.



FIG. 13.



FIG. 14.



FIG. 15.



FIG. 16.



FIG. 17.

Twenty-fifth day. L. Tail is separated from body by a clear space ; granules at site of one of the upper figures (Fig. 15).

Six weeks. L. Only three small figures with rounded outlines seen (Fig. 16).

Two months. L. Posterior figures smaller (Fig. 17).

Three months. L. Lens quite clear.

The injury in this case was probably due to concussion.

CASE 14.—Wm. U. (Mr. Morton), æt. 19.

1.6.08. Four hours before admission patient was struck in L. by a small piece of copper.

State on admission :—

L. Perforating wound of cornea below with many glistening particles between the lips. Small foreign bodies and piece of entangled iris removed from wound. Skiagraphy shows no foreign body.

Fourteenth day. L. One large and one small figure at centre of back of lens like thin bubbles with crenated edges; no anterior opacity. L. V. = 6/18 partly.

One month. L. No change in lens opacity.

Five months. L. Posterior opacity of the same shape as before, but very faint, and apparently of dusty consistence. Two fairly large, and one or two small, vacuoles in lower outer part of lens. One small dense anterior opacity in the lens opposite to the inner end of the coloboma, with faint traction folds passing from it. V. $\bar{c}$ + 2 Dcyl. V. = 6/18.

Eighteen months. L. Granular posterior opacity below, and to a much less extent centrally, seen by oblique illumination as grey chagrin in the lines of the lens fibres; a very few small vacuoles. Anterior opacity as before.

$$\text{V. } \bar{c}. \frac{+0.5 \text{ Ds.}}{-2 \text{ Dcyl. } 30^\circ \text{ down and in}} = 6/18.$$

CASE 15.—Albert K. (Mr. Lawson), æt. 17.

21.9.08. L. Injured by a fragment of steel five days ago.

State on admission :—

L. Small perforating scleral wound 3 mm. internal to limbus. Lens clear and *in situ*. Foreign body in vitreous. V. $\bar{c}$ + 1 Ds. = 6/12.

24.9.08. Foreign body drawn into A. C. and removed with iridectomy.

27.9.08. L. Small discoid opacity composed of fine granules at centre of posterior aspect of lens.

8.10.08. L. Lens clear.

$$\text{V. } \bar{c}. \frac{-.75 \text{ Ds.}}{+2 \text{ Dcyl. } 30^\circ \text{ down and out}} = 6/9.$$

GROUP 4.—*Case in which the Lens Changes were perhaps very slowly progressive.*

CASE 16.—Chas. H. (Mr. Collins), æt. 31.

6.5.07. Struck in R. four days ago while cutting down a hoof with a sword.

State on admission :—

R. Lids swollen ; sticky conjunctival discharge. Cornea slightly hazy ; scar at inner margin. Aqueous somewhat turbid ; fluid hypopyon. Iris fairly bright ; scar in it opposite corneal scar. V. = p.l.

9.5.07. Foreign body removed through scleral incision below.

17.5.07. R. Much injection ; two or three post-corneal deposits, much coarse vitreous opacity, and flocculent grey mass at bottom of vitreous.

23.5.07. R. Many fine light brown post-corneal deposits. Small circumscribed opacity in lens opposite to wound.

27.5.07. R. Post-corneal deposits rather grosser, but less numerous. V. = 6/18.

Three months later. R. No post-corneal deposits. Iris bright and pupil active. Minute feathery opacity at anterior, and rather more extensive opacity at posterior, surface of lens, taking origin apparently in the region of the original wound. Vitreous is fairly clear, but there are a few rather gross freely floating opacities, and some grey streamers from the ciliary region below. Central region of fundus normal, except for a break in the outline of the outer margin of the O.D. Fairly extensive superficial choroido-retinal atrophy below. V. = 6/9 partly, and J 2 with difficulty.

Could not be traced further.

GROUP 5.—*Cases Exhibiting Equatorial Changes for the most part in the form of a Vacuolar Palisade. Course of Changes in Lens Progressive.*

CASE 17. Henry F. (Mr. Flemming), æt. 22.

3.2.08. L. Injured by fragment of iron three months ago.

State on admission :—

L. Small scar at lower margin of cornea. In lower part of

lens is a small, dense, anterior opacity ; posterior lens opacity. Foreign body in vitreous. V. = 6/36.

4.2.08. Foreign body removed through corneal wound.

14.2.08. L. Posterior opacity of indefinite structure or granular. V. = 6/36.

30.3.08. L. Posterior opacity for the most part granular ; some vacuoles. V. = 6/18.

21.4.08. L.V. = 6/12.

One year. L. Considerable opacity at anterior surface of lens containing some rusty brown pigment. Posterior opacity less below, but elsewhere granules are more evident. Vacuoles midway between centre and periphery, for the most part discrete, some of which have appeared since last note. V = 6/18.

Two years. L. Anterior opacity has increased. Much fine vacuolar and granular posterior opacity. Palisade of peripheral vacuoles, many of them discrete, above and to a less extent on each side. V. = 6/60.

CASE 18.—Chas. N. (Mr. Parsons), æt. 15.

10.9.07. R. Injured by a piece of wire four days ago.

State on admission :—

R. Perforating scar in upper part of cornea. Rent in anterior lens capsule ; neighbouring lens very slightly swollen. Posterior lacework opacity with festooned border. V. = 6/60.



FIG. 18.



FIG. 19.

R. Posterior opacity became gradually less dense. The condition six weeks later is represented in Fig. 18. (The thick dark line in this figure represents the scar in the anterior capsule.)

Two months. Central part of posterior opacity almost clear. V $\bar{c}$ + 2 Ds = 6/36 partly.

Three and a half months. R. Posterior opacity is represented only by granules very scantily disposed in a star-shaped figure. A peripheral palisade opacity has developed below, composed of lines with a radial arrangement forming a fine, close network centrally, and a coarser pattern peripherally. (Fig. 19.)

Six months. Diffuse interstitial opacity with shifting grey reflex under oblique illumination. V. = hand movements.

Lens removed.

CASE 19.—Henry P. (Mr. Collins), æt. 47.

20.1.08. Injured in L. while hammering iron six weeks ago.

State on admission :—

L. Perforating scar in lower part of cornea. Broad posterior iris synechia behind corneal scar ; neighbouring lens opaque, flocculent, and protruding. Indefinite central interstitial haze of lens. Posterior opacity with festooned border, both vacuolar and granular. Peripheral lens opacity composed of vacuoles, few and discrete above, in dense palisade below. At level of posterior opacity, and opposite anterior wound, is a horizontal slit-like figure with grey opacity at upper and lower margins, strongly suggestive of a rent in posterior capsule. V. = 2/60. Skiagraphy shows no foreign body.

25.3.08. L. Posterior peripheral vacuolar and interstitial opacities of lens are more extensive.

Nine months. L. Lens opaque throughout. V. = p.l.

Lens removed.

CASE 20.—Wm. A. (Mr. Lang), æt. 32.

30.1.07. Immediately before admission was struck in L. by a piece of glass lamp shade.

State on admission :—

L. Perforating corneo-scleral wound. Superficial wound of lens. Blood in vitreous.

31.1.07. L. Iridectomy.

12.2.07. L. Feathery posterior lens opacity. V. = 6/36.

19.2.08. L. Shifting grey reflex in all parts of lens opposite scar. Grey satin-like striated opacity throughout whole posterior surface of lens, seen by oblique illumination, but not with transmitted light, which reveals only central granular opacity and small segments of peripheral vacuolar palisade.

Lens removed.

CASE 21.—Frank B. (Mr. Collins), æt. 18.

29.3.06. R. Struck by a piece of steel three days ago.

State on admission :—

R. Perforating scar near centre of cornea. Star-shaped posterior lens opacity; some external cortical opacities. Foreign body in vitreous.

Foreign body removed through scleral incision.

9.4.06. R. Lens opacities more marked.

31.5.06. R. Counts fingers.

GROUP 6.—*Cases in which Opacity Developed with Varying Rapidity.*

CASE 22.—Wm. P. (Mr. Lawson), æt. 14.

6.4.08. R. Struck to-day by a piece of board.

Condition on admission :—

R. Large perforating wound in inner part of cornea. Anterior surface of lens wounded; star-shaped opacity with seven rays at posterior aspect of lens, lacework type.

Three weeks. R. Posterior opacity is larger and more discoid; a process passes in from it towards the lens margin. Central part is clearest.

Five weeks. R. Posterior opacity less definite in outline, and decidedly less dense, especially centrally.

Seven weeks. R. Posterior opacity much less, and consists of a halo of lacework opacity surrounding a fairly clear central region in which only granules are seen. V. = 6/36.

Six months. R. Lens has been reduced to a thin shell. Posterior opacity is represented by coarse, craggy, radial opacities at the site of the lacework opacity last seen, together with a good deal of irregular granular opacity and some vacuoles.

CASE 23.—Arthur W. (Mr. Flemming), æt. 30.

31.7.08. Injury to L. five days ago with a pin.

Condition on admission :—

L. Perforating corneal wound just inside outer part of limbus. Small anterior lens opacity; dense stellate posterior opacity. V. = hand movements.

Sixteen days. L. Anterior opacity in the form of groups of

radially disposed striæ; posterior opacity has the appearance of a dense irregular interlacement. V. = fingers.

Twenty-three days. L. Anterior opacity composed of fine radial lines; posterior opacity appears to consist of vacuoles.

Six weeks. L. Anterior opacity granular, posterior opacity composed of vacuoles for the most part discrete.

Six months. L. Lens completely opaque.

Lens removed.

CASE 24.—Wm. M. (Mr. Morton), æt. 20.

17.10.07. L. Struck to-day by a large flying fragment of steel.

State on admission :—

L. Corneal wound; iris prolapsed. Lens considerably lacerated.

L. Iridectomy of prolapsed iris.

23.10.07. L. Posterior lacework lens opacity extending to margins. Some anterior vacuolar opacity at site of injury.

11.11.07. L. Clear zone between central part of posterior opacity and periphery. V. $\bar{c} + 2$ Ds. = 6/36 partly.

One year. L. Lens shrunk and flat. Much anterior opacity like scattered wet sand; rather dense irregular granular posterior opacity extending nearly to periphery. Some interstitial opacity. V. = fingers, not improved.

CASE 25.—George I. (Mr. Flemming), æt. 32.

16.9.07. Struck in L. 26 days ago while drilling iron.

Condition on admission :—

L. Perforating scar in lower part of cornea, behind which is a rent in anterior capsule from which projects flocculent lens matter; fleecy opacity of neighbouring lens. Posterior lens opacity. V. = hand movements. No foreign body shown by skiagraphy.

One month. L. Thickening of anterior capsule around rent. Definite lacework posterior opacity, which seems somewhat less dense than at last note. Some indefinite interstitial opacity. V = fingers.

Three months. L. Lens completely opaque.

Lens removed.

CASE 26.—Ernest L. (Mr. Morton), æt. 21.

14.10.07. L. Injured with a large piece of iron to-day.

State on admission :—

L. Perforating wound at limbus above. Knuckle of iris forward to it. Lens appears clear. V. = 6/36.

One day. L. Star-shaped posterior lens opacity.

Three days. L. Posterior lens opacity has lacework pattern.

Deep interstitial lens opacity above. V. = fingers.

Nine days. L. Posterior opacity appears rather less ; interstitial opacity denser. V. = 6/60.

Interstitial opacity increased and lens was needled.

CASE 27.—Chas. H. (Mr. Gunn), æt. 32.

18.5.06. L. Injured to-day by a flying fragment of steel.

State on admission :—

L. Perforating wound at inner margin of cornea. Prolapse of iris.

Foreign body and prolapsed iris removed.

Two weeks. L. Posterior lacework lens opacity ; lens wounded internally.

Seven months. L. Lens completely opaque.

CASE 28.—Geo. C. (Mr. Parsons), æt. 24.

1.7.08. R. Struck by a piece of rivet two days ago.

State on admission :—

R. Linear wound in cornea to outer side of centre. Anterior lens opacity ; posterior opacity consisting of 10 lacework leaflets, each of which is fringed with a border of vacuoles like a string of pearls. V. c. + 1.5 Ds. = 6/12 partly.

8.7.08. R. Posterior opacity much increased and spreading out to periphery of lens ; anterior opacity also larger. V. = fingers.

Four months. R. Lens reduced to a thin shell. Dense posterior opacity with festooned border, components of opacity showing a roughly radial arrangement.

GROUP 7.—*Concussion Injuries.*

CASE 29.—Edward O. (Mr. Lang), æt. 13.

25.2.06. R. Struck by an arrow yesterday afternoon.

Nine days later opacity seen at posterior aspect of R. lens.

Fine vitreous opacity and œdema of retina.

17.9.06. R. V. $\bar{c}$. + 3 Ds. = 6/24. Could not be traced further.

CASE 30.—Arthur E. (Mr. Lang), æt. 17.

25.2.06. Was struck in L. three hours ago by the hook of a Sandow's accumulator. Lens dislocated inwards, and has striate posterior cortical opacities.

Two years and eight months. L. Irregularly granular rather faint opacity at anterior and posterior surface of lens, extending from equator to beyond centre.

V. P. + 1 Deyl. 75° down and in = 6/9.

CASE 31.—Desmond S. (Mr. Lang), æt. 27.

14.12.07. L. Struck by a whip seven days ago.

Condition on admission :—

L. Lens tremulous ; striate posterior opacity in the lines of the lens fibres. Two ruptures in iris.

Lens became rapidly opaque and was removed.

CASE 32.—Louisa T. (Mr. Morton), æt. 11.

24.9.06. Struck in R. two days ago by a dart from an air-gun. Hyphæma ; traumatic mydriasis. No perforation.

Two days. R. Irregular posterior lens opacities. Coarse vitreous opacities.

Two weeks. R. Lens opacity extending.

Five weeks. R. Lens almost opaque.

Lens removed.

GROUP 8.—*Further Examples of late Appearances of Posterior Traumatic Cataract.*

CASE 33.—Arthur P. (Mr. Worth), æt. 35.

18.4.07. L. Injured two days ago by a flying fragment of steel. Foreign body sticking in lens.

Foreign body removed through corneal incision.

Six days. L. Lens slightly tremulous ; small localised opacity at site of injury. No posterior opacity. V. $\bar{c}$.

+ 2.5 Ds.
+ 0.75 Deyl. V = 6/9 partly.

Eighteen months. L. Small grey dense anterior opacity in

lens; three vacuoles in region of posterior surface. No other opacity. V. $\bar{c}$. + 1.75 Deyl. H = 6/6 partly.

CASE 34.—Jas. S. (Mr. Morton), æt. 63.

8.10.06. L. Struck yesterday by a flying fragment of steel.

State on admission :—

L. Wound in upper outer part of cornea; hole in iris behind it. Opacity in lens radiating from region of wound. V. $\bar{c}$. + 3.5 Ds. = 6/24.

9.10.06. Foreign body drawn into A.C. and removed through corneal incision.

17.10.06. L. Lens opacity as on admission with some vacuolation in upper and outer part. V. $\bar{c}$. + 3.5 Ds. = 6/36.

Two years. L. Small dense lens opacity at outer equator passing at once into posterior horizontal band of granular opacities which become very fine when traced inwards. Small central posterior opacity composed of granules and one vacuole. No anterior opacity. V. $\bar{c}$. + 3.5 Ds. = 6/9.

CASE 35.—Henry O. (Mr. Lawson), æt. 17.

26.10.07. R. Struck by a stone 14 years ago; sight of R. defective since accident.

State on admission :—

R. Small, flat, rosette-shaped, irregular pattern, anterior lens opacity. Similar posterior lens opacity. V = 2/60.

Lens removed.

GROUP 9.—*Illustrative of the Effect of Reopening a Wound in the Lens.*

CASE 36.—John A. (Mr. Gunn), æt. 39.

11.1.07. L. Struck by a fragment of iron eight months ago. Sight misty three weeks.

State on admission :—

L. Horizontal scar in upper part of cornea. Feathery opacity at posterior, and to a less extent at anterior, surface of lens. In the upper part of the lens towards the posterior surface is a shining particle of steel. V. = 6/60. No siderosis.

12.1.07. Foreign body brought into anterior chamber and extracted through a corneal incision.

Very slow reaction in lens with some swelling. Lens eventually removed.

CASE 37.—Alfred A. (Mr. Morton), æt. 35.

18.3.07. L. Injured by a fragment of steel yesterday.

Condition on admission :—

L. Horizontal perforating wound in upper part of cornea. Iris lacerated. Bright foreign body in clear lens.

19.3.07. Foreign body removed through original wound. A mass of lymph accompanied it.

21.3.07. L. Hypopyon.

3.4.07. L. Quieting. Some grey post-corneal deposits. Lens opacity at site of injury more extensive. Posterior stellate lacework opacity.

L. Remained irritable. Pupil became occluded by inflammatory exudate and lens opacity increased. Eye removed seven months later.

CASE 38.—Geo. H. (Mr. Lang), æt. 51.

1.6.08. Struck in L. nine days ago by fragment of steel.

State on admission :—

L. Perforating corneal scar. Small grey scar in lens capsule, behind which is lustrous foreign body in perfectly clear lens.

Foreign body removed through corneal incision.

2.6.08. L. Lens opaque throughout.

7.6.08. L. Lens swollen ; acute glaucoma. Lens removed.

GROUP 10.—*Unclassified Cases of Posterior Traumatic Cataract.*

CASE 39.—John C. (Mr. Collins), æt. 49.

15.4.07. Struck in L. while using a pickaxe six days ago.

State on admission :—

L. Lens injured to outer side ; posterior polar opacity.

Foreign body removed through scleral incision.

Twelve days. L. Lens opacity has not spread. V. = 6/60.

Could not be traced.

CASE 40.—Edward G. (Mr. Marshall), æt. 33.

11.1.06. Struck in L. to-day by a flying fragment while using a hammer and chisel.

State on admission:—

L. Perforating wound just outside limbus. Feathery opacity of lens extending from neighbourhood of wound. V. = 6/18.

22.1.06. Foreign body removed from vitreous through scleral incision.

2.2.06. L. Commencing posterior lens opacity. Exudate in lower part of vitreous and detachment of retina. V. = 6/36.

CASE 41.—Wm. M. (Mr. Spicer), æt. 14.

24.10.07. R. Struck by a piece of wire six weeks ago.

Condition on admission:—

R. Perforating corneal scar. Anterior lens opacity opposite to scar passing deeply into lens. Posterior lacework opacity with festooned border. V. = fingers.

Lens needled.

CASE 42.—Arthur L. (Mr. Collins), æt. 43.

9.5.07. L. Struck by a flying fragment of steel four days ago.

State on admission:—

L. Corneo-scleral wound. Opaque track through lens. Foreign body could not be removed.

Twenty-three days. Posterior part of lens quite opaque. Lens became opaque throughout, and had been almost completely absorbed after a year.

CASE 43.—John N. (Mr. Worth), æt. 1½.

7.9.08. Perforating wound of R. cornea with hat-pin two days ago.

Fourteen days. R. Anterior lens opacity below, and posterior cortical opacity.

Lens became opaque throughout.

CASE 44.—Albert B. (Mr. Parsons), æt. 28.

13.8.07. R. Injured with a scarf-pin three months ago.

R. Scar in lower and inner part of cornea. Opacity

throughout lens, with dense star-shaped posterior opacity.
V. c. + 2.75 Ds. = 6/24.

Lens removed.

SUMMARY.

Character of opacity when first seen. (The bracketed figures refer to the case numbers, and the periods of time to the intervals between dates of accident and first observation):—

Striæ: (16) 3 hours, (30) 3 hours, (1) 6 hours, (6) 6 hours, (2) 1 day, (40) 1 day, (31) 7 days, (20) 13 days, (7) 17 days, (36) 8 months.

Striæ with some vacuoles between them: (3) 1 day, (8) 5 days.

Lacework: (22) 6 hours, (5) 2 days, (4) 3 days, (18) 4 days, (23) 5 days, (24) 6 days, (27) 14 days, (37) 16 days, (25) 26 days, (9) 6 weeks, (41) 6 weeks.

Lacework fringed with vacuoles: (28) 2 days.

Vacuoles: (13) 11 days, (14) 14 days, (10) 17 days, (33) 18 months.

Granules: (15) 6 days.

Striæ, vacuoles, and granules: (11) 9 weeks.

Vacuoles and granules: (12) 13 days, (19) 6 weeks.

Unclassified: (26) 1 day, (34) 1 day, (43) 2 days, (32) 2 days, (21) 3 days, (42) 4 days, (39) 6 days, (29) 9 days, (17) 3 months, (44) 3 months, (35) 14 years.

Changes observed:—

Vacuoles appearing between striæ: (2).

Vacuoles appearing in mid-ribs: (2), (3).

Striæ to lacework: (2), (11).

Striæ to vacuoles: (3), (7), (8).

Striæ to vacuoles and granules: (6), (11), (20).

Striæ to granules: (11), (43).

Lacework becoming less extensive: (5), (18), (24).

Lacework becoming less dense: (7), (18), (22), (25).

Lacework to vacuoles: (2), (4), (5), (23).

Lacework to vacuoles and granules: (7), (9).

Lacework to granules: (18), (22), (24).

Vacuoles becoming larger: (5), (11).

Vacuoles becoming discrete: (23).

Vacuoles becoming larger, less numerous, and discrete:
(2), (3), (4), (7), (10).

Larger vacuoles breaking up into smaller: (13).

Vacuoles becoming smaller: (13).

Vacuoles to granules: (2), (3), (6), (7), (8), (10), (12),
(14).

Vacuoles disappearing: (2), (8), (13).

Granules disappearing: (2), (15).

Final result:—

Lens clear: (13), (15).

Granules: (2) 1 month, (6) 1 month, (3) 13 weeks, (24)
1 year, (30) $2\frac{3}{4}$ years.

Granules and vacuoles: (19) 6 weeks, (12) 5 months,
(18) 6 months, (8) $1\frac{1}{3}$ years, (11) $1\frac{1}{3}$ years, (14)
 $1\frac{1}{2}$ years, (10) $1\frac{1}{2}$ years, (17) 2 years, (34) 2 years,
(7) $2\frac{1}{4}$ years, (9) 3 years.

Vacuoles: (23) 6 weeks, (33) 18 months.

Lacework: (41) 6 weeks.

Indefinite: (35) 14 years.

General lens opacity developing: (1), (4), (5), (26)
3 days, (20) 19 days, (32) 2 weeks, (21) 3 weeks,
(31) $3\frac{1}{2}$ weeks, (19) 6 weeks, (43) 6 weeks, (25)
3 months, (44) 3 months, (23) 6 months, (18) $6\frac{1}{2}$
months, (37) 8 months, (17) 2 years.

Shrunken lens: (28) 4 months, (22) 5 months, (42)
1 year.

Not traced: (39), (40).

REMARKS.

From a study and comparison of the changes observed in the various cases described above, it seems permissible to consider the following series of phenomena as the typical and characteristic expression of the succession of changes occurring in a posterior traumatic cataract. Within a few hours of

the receipt of injury there appear in the region of the posterior surface of the lens feather-like opacities, whose precise orientation is dependent upon the architecture of the lens fibres in relation to the lens star, and exhibits accordingly some variations in individual cases. These feather-like opacities are first seen in the central region, whence they tend to extend outwards towards the periphery of the lens, to become continuous with a fringe of short, radial striæ disposed in conformity with the long axis of the equatorial fibres, and also appearing at an early date.

At a slightly later date vacuoles may, in some cases, be observed in the mid-ribs of the feather-like opacities, between their rays, and between the peripheral striæ. Usually within the next few days the character of the opacity changes, the striate formation being replaced by a rather dense lacework pattern, in which, as a rule, few definite vacuoles can be recognised with certainty, but in which the radial plan may still be traced. This in turn is succeeded by a definitely vacuolar pattern, and at this stage not infrequently rather large vacuoles, either discrete or in a palisade formation, and having their axes in conformity with that of the lens fibres, may be observed in the equatorial region of the lens. The vacuolar pattern next becomes more open, as though from the formation of larger vacuoles by the coalescence of smaller ones.

In the later stages the central figure becomes broken up into discrete vacuoles, which become progressively smaller in size, until finally all that remains visible is a collection of fine granules, which may by their arrangement afford some suggestion of the antecedent stellate figure.

Various hypotheses have been advanced to explain the peculiar character and behaviour of the posterior traumatic cataract illustrated by the above cases. Twenty years ago Schlösser (*Experimentelle Studie über traumatische Kataract*, 1887), referring to a case seen clinically, concluded that the opacity not improbably owed its existence and character to a distension of preformed lymph spaces, the existence of

which in the lens of the rabbit he considered to be demonstrated by his experimental studies. Fuchs, at about the same time (*Über traumatische Linsentrübung*, Wiener klin. Wochenschr., 1888), described and commented on 11 cases of posterior traumatic cataract. In one case the striate opacity developing at the posterior surface of the lens, in a case of injury at the equator, was noticed to disappear, except in the neighbourhood of the wound, where it later assumed a lacework, and finally a granular character. In this region also two or three rows of peripheral loops, parallel with the margin of the lens, were noticed in the later stages. Fuchs accepted Schlösser's conclusions as to the existence of a system of physiological lymph spaces in the lens, the distension of which with opaque material he considered to be responsible for the lacework and loop-like figures observed. More recently, zur Nedden (*Zeitschrift für Augenheilkunde*, vol. ii, p. 389, 1904) has suggested that the changes under consideration are due to a shifting of the lens within its capsule, whereby the connection with the posterior capsule becomes loosened or severed. As a result of this separation, fluid is able to gain access to the cement substance between the fibres, and to cause their separation.

If an attempt be made to explain the appearances observed in the cases here described on one of the above hypotheses, it will be evident that a supposition which would attribute the central lacework patterns and palisade of loops to the distension of a system of lymph channels, becomes at once untenable when the various modifications of these figures, resulting in the later stages in an appearance of discrete vacuoles, is borne in mind. If further evidence were required, it would be only necessary to refer to the researches of Leber, which render the existence of lymph channels, either in the lens capsule or between the fibres, in the highest degree improbable.

Against the theory that the peculiar patterns are due to a collection of fluid between the lens fibres, the following arguments may be urged. In the early stages of striate

opacity, limited to a small area of the posterior part of the lens, the opacity exhibits no peripheral boundary-line (roughly concentric with the lens equator) such as would surely be expected, unless it be supposed that the fluid at this stage lies only in the antero-posterior planes between the lens fibres. The latter supposition appears untenable; for it would seem to involve the further assumption that, in the lacework patterns developing from the striation, it is the dark lines, and not the clear areas, which are the site of the fluid, and this in turn would lead to the ridiculous conclusion that the apparent discrete vacuoles observed in the later stages are not really vacuoles, but appearances produced by the persistence between the lens fibres of intensely thin rings of fluid. On the supposition that the opacities are due to fluid of the nature of lymph, lying in all planes between the lens fibres, even if the before-mentioned difficulty presented by the absence of any line of limitation in the earliest stages be overlooked, the existence in the lacework patterns of fine lines between the individual vacuoles, and the comparatively slow coalescence of the latter, and finally the very protracted persistence of some of the vacuoles, are not easy to explain. The final granular appearances might, however, perhaps be attributed to albuminous deposits from the pre-existing fluid.

If, however, it be assumed that the essential factor in the production of the phenomena under consideration is a swelling of the actual lens fibres which may be preliminary to further changes, as a result of injury, the above-mentioned difficulties disappear. The dark lines seen by transmitted light in the stage of striation then become intelligible, as the result of the different conditions of refraction of light through and between the contiguous swollen posterior lens fibres. It is conceivable that such changes, limited to mere swelling of the fibres, may be capable of retrogression without permanent damage to the tissue. The lacework patterns and peripheral palisades would result from irregular swelling of the fibres and coincident alteration in the constitution of

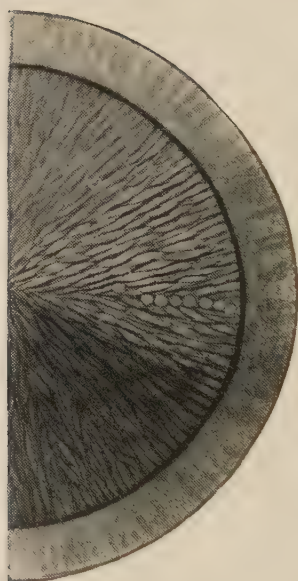


FIG. 20.—Oblique Illumination.

their protoplasm ; while the development of the more open-work patterns would be attributable to the coalescence of contiguous masses of fluid protoplasm, owing to the breaking down of the intervening fibre sheaths and cement substance. The later stages of discrete vacuoles would result from a subsequent partial absorption of this fluid, while the final stages of vacuoles and granules would be explicable as the result of permanent changes in the residual lens fibres and the deposition of products of degeneration during the process of fluid absorption. The vacuoles sometimes seen in the early stages in the mid-ribs of the figures might be assumed to owe their origin to fluid which has exuded from the abutting ends of the affected lens fibres, and some rôle might be assigned to the presence of such fluid amongst the lens fibres in the production of the figures formed subsequently. The linear equatorial opacities concentric with the lens margin, noted by Fuchs and others, may be explained as an effect of refraction between the unaltered central fibres and the affected equatorial fibres, or as due to an actual breaking down of the latter. The fact that changes are most frequently observed at the posterior aspect and equator of the lens may perhaps owe its explanation to the circumstances that the posterior capsule is considerably thinner and less resistant than the anterior, and is therefore less likely to offer opposition to swelling of the lens fibres, while the equatorial fibres, being young and containing relatively more fluid protoplasm, react more readily to disturbing influences.

This supposition, that the changes at the posterior aspect of the lens are due to actual swelling of the lens fibres themselves, is strengthened by the fact that it is possible in some cases of lens injury to detect changes in the lens beneath the anterior capsule which are strongly suggestive of swelling of the lens fibres. Thus in the case represented in Fig. 20, Pl. XI, in which the lens had been injured 14 days previously by a piece of steel which could be seen lying in its substance, the whole of the inner half of the anterior

surface of the lens could be seen to be divided up into small areas, for the most part resembling the small spindles seen in the fruit of an orange, and elongated in the direction of the course of the lens fibres. These areas presented a flat surface, which appeared according to the direction of incidence of oblique light to be either translucent like dark ice or covered by an opaque fine chagrin, and were separated from one another by linear depressions which appeared black to both oblique and transmitted light. A similar, although much less extensive and obvious, change was observed three days after the needling of a lens for high myopia (Reginald C., Mr. Collins, Reg. No. 650, 1909), and also in a case of perforating wound of the lens (John F., Mr. Fisher, Reg. No. 1939, 1909). In the latter case the condition was apparently responsible for several peripheral anterior opacities of sectorial shape with apex to centre which were visible, however, only to transmitted light, under which they exhibited an irregularly lacework pattern. These opacities had completely disappeared five days after the injury, with the exception of one in the immediate neighbourhood of the wound, which still persists, five weeks after the injury, as a rather more open lacework corresponding to the margins of elevations of swollen clear lens substance.

In further support of the theory that the posterior changes are due to changes in the lens fibres, it is to be noted that the impression obtained by examination with transmitted light of the existence of bubbles of fluid amongst the lens fibres is not reinforced by the use of oblique illumination. Under this method of examination the opacities in the lacework patterns appear as rather woolly, or slightly lustrous, lines of considerable thickness, the opacity fading gradually towards the centres of the clear areas, and not as thin highly refracting lines such as would be expected between bubbles of lymph-like fluid. In some cases, again, it is difficult to reconcile the bizarre figures seen by transmitted light with the view that the appearances are

due to fluid amongst the lens fibres. This is well exemplified by Fig. 8 (Case 10), where, however, the splinter-like formations and distorted figures become intelligible when regarded as altered lens fibres. Finally it may be stated that the results of experiments carried out by the author, and shortly to be published elsewhere, afford support to the view that the essential changes concerned in the production of the posterior opacities occur in the lens fibres.

Fuchs has expressed the opinion that if the development and clearing of posterior traumatic cataract were due to destructive changes and absorption of lens fibres it should be possible to demonstrate during retrogression of the changes an increasing hypermetropia or hypermetropic astigmatism. This is a point on which it is difficult to obtain definite evidence from the cases described above, since it is impossible to disregard or estimate the possible changes in the state of refraction of the injured eye dependent on the associated injury of structures other than the lens. In Case 9, however, the findings were suggestive of slight progressive change in the direction of hypermetropia, while in another case (John F., Reg. No. 1939, 1909), at present under the care of Mr. Fisher, the affected eye showed five days after the removal of a foreign body from the vitreous through a corneal incision 4 D, 15 days later 5 D, and 14 days later 6 D, of hypermetropia: the refraction of the fellow eye shows 2.5 Ds., 1 Dc. 75° down and out of compound hypermetropic astigmatism. The injured eye is the subject of posterior traumatic cataract, which is at present undergoing slow retrogressive changes centrally.

It seems probable that direct injury or concussion of the lens tissues, rather than direct access of the aqueous humour, should be held responsible for the production of the posterior opacities; for on the one hand there is good evidence that perforation of the lens capsule is not essential for the production of posterior traumatic cataract, and on the other perforation, although frequently, is not invariably, associated with posterior opacity. The reopening of an old

wound in the capsule is, however, very liable to be followed by complete opacification of the lens. Both of these latter features are well exemplified by the two cases cited above, in which the removal of a piece of steel embedded in a clear lens was followed by a rapidly developing cataract. The explanation of the course pursued in these cases probably lies in the fact that the bursting force exerted during the exit of the foreign body would be likely to produce a larger lesion of the anterior capsule than would the incising force exercised on entry, while the probability of an immediate second union of the reopened capsule would be extremely remote.

The prognosis, both immediate and remote, in a case of traumatic cataract should always be guarded. In the early stages it is difficult to say whether improvement will occur, or, if it does occur, will be maintained. Mere improvement in vision is not to be regarded as a safe criterion in affording a favourable prognosis, for clearing of central opacities may be associated with progressive changes at the equator of the lens. On this account especial attention in examination should always be given to the presence and behaviour of changes at the lens periphery, more especially as the mere presence of these is probably a feature of unfavourable import. In every case a lens which has been affected by posterior traumatic cataract is to be regarded as a damaged tissue abnormally susceptible to deleterious influences.